The New England
Journal of Medicine

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Volume 233 DECEMBER 13, 1945 Number 24

# OCCUPATIONAL DERMATOSES: DISABILITY AND COMPENSATION*

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DISEASES of the skin comprise 65 per cent of all occupational disease. There is, however, surprisingly little material indicating the actual amount of disability that occurs in these cases, the expense in terms of compensation paid or the medical costs involved. Compensation laws have been enacted in almost all the states, and it can be expected that such studies will be available later. These laws and the progress made since the original compensation act passed in Wisconsin in 1911 have been discussed by Foerster.[1] He points out that within fourteen years there occurred in that state three times as many compensable cases in the last year of the period as in the first year. Furthermore, in the last year, the expenditure for indemnity had increased six times, and the medical expenditures ten times. It is reasonable to suppose that occupational dermatoses have increased in a like measure, with similar increases in disability and compensation figures.

Industrial accidents are, of course, far more numerous than occupational diseases and provide many more serious cases. The study of these surgical cases has led to the development of effective methods of prevention and treatment, whereas occupational diseases, with their large proportion of cutaneous cases, have been relatively neglected. True, great advances have been made in the control of lead poisoning, silicosis and a few other serious conditions. On the other hand, the toll that cutaneous disturbances take in the occupational field is large and deserves more study than it has thus far received.

A search has been made for data that support this statement of losses, but there is to be found in the medical literature little evidence of the actual losses by disability, and only a few statements of the costs in connection with cases of occupational dermatoses. Insurance companies and state accident commissions do not have readily available the data on these items. Some commissions in their annual reports present figures, but this occurs in only a few states, and no commission can register all the cases occurring in its state.

An attempt has been made to obtain recent figures concerning four items in connection with cases of disability due to occupational cutaneous disease: compensation paid during disability, medical expenses, amount of wages lost and the duration of disability or of time lost from work. The purpose of this paper is to present the data thus obtained and to discuss, with the hope of eventually reducing such losses, the criteria for the diagnosis of occupational dermatoses and the contributing factors of long continued disability.

Disability compensation and medical expenses, together with lump-sum settlements and the like, represent the total cost, but they do not include the wages lost. This loss is to a certain extent offset by the compensation, but the latter alone does not maintain the worker and his family as before, and such loss to the family is difficult to calculate. The time lost by a skilled worker also often represents a real loss to the employer, which is difficult to measure in dollars and cents.

In reviewing these data, it is recognized that a large number of workers with occupational dermatoses have no disability, or disability for an extremely short period. The number of these minor cases and the duration of disability of the major cases could be substantially lessened through better methods of early detection, correct diagnosis and insistence on adequate and proper treatment. This would obviate a large amount of time now lost from overtreatment and from secondary infection.

In contrast to the minor cases there occur from time to time long-drawn-out disabilities that are extremely costly to the employer, the employee and the insurance company. Blaisdell[2] cites 3 of these. In one, a shoe stitcher with minor disturbances on the hands was disabled for sixty weeks. The total compensation was $567.00, the medical fees were $112.00, and the total cost to the insurer was $864.00. In another case, a shoe laster received for ninety-six weeks of total disability and thirty-four weeks of partial disability compensation totaling $1902.57 and a lump-sum settlement of $457.00. Medical

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care cost $165.00, and the cost to the insurer was $2680.00. In the third case, a shoe ironer with a constantly recurring dermatitis received for nearly seven and a half years a weekly compensation of $11.83. The payment automatically stopped when they reached the full amount allowed by the law, $4500. Medical care and other costs amounted to $1000. There could well be some doubt, at least from the dermatologic standpoint regarding the validity of some of the recurrences suffered by this patient.

In a case from another source, a disability of more than forty-eight weeks resulted from a dermatitis and its complications, with total medical expenses of $565.00 and a total compensation cost, including a lump-sum settlement, of $1588.00. The wages lost were $864.00.

The industrial-accident commissions of three states, Wisconsin, New York and Ohio, have been able to furnish data on the losses in protracted disability cases. The recent reports of the Industrial Commission of Wisconsin (Table 1) are of interest in connection with the disability and compensation from dermatitis. The reports also show that the number of cases of dermatitis increased from 251 in 1940 to 775 in 1943, with cases resulting from exposure to oils and lubricants rising from 15 in 1940 to 161 in 1943. The average number of days lost and the average indemnity and cost of medical care from 1937 to 1942 are shown in Table 2. This table shows a creditable tendency in the face of what was probably an increasing number of cases due to a larger number of employees.

A recent report from the New York State Department of Labor states that 1700 cases of occupational dermatitis were adjusted in 1943. These patients were awarded a total of 14,605 weeks of compensation, with an average of 9.2 weeks (Table 3). The total compensation awarded was $222,311.00, with an average of $131.00. (It would be interesting to know whether the costs in Wisconsin and New York were computed on the same basis.) If the lost time in the temporary-disability group is increased by that in the cases with less than thirty-five days lost, in which no compensation was paid for the first week, the total amounts to 13,311 weeks. The wage loss in this group of cases has been estimated as $160,000. Information concerning the amount of medical expenses incurred is not available.

A report by the Industrial Commission of Ohio gives no information regarding compensation paid or the medical expenses of occupational dermatoses, but furnishes data concerning the time lost in 1943. Of 4711 cases encountered during that year, 2 of which had a fatal outcome and 1 permanent partial disability, there was disability of over seven days in 780 and of seven days or less in 370; no time was lost in 3558. The total lost time was 32,535 days, of which 12,000 were incurred in the 2 fatal cases and 600 in the case with permanent partial disability. Eighteen thousand, three hundred and seventy-three days were lost in periods over seven days and 1562 days in those of seven days or less. Dust, liquids and oils accounted for a large part of the disability.

Through the co-operation of the American Mutual Liability Insurance Company (Table 4), it has been possible to review the claims in cases of dermatitis occurring during the last five years. With the figures for 1940 and 1944 incomplete, 753 cases were reviewed with regard to compensa-

TABLE 1. *Disability and Compensation for Dermatitis in Wisconsin (1941 and 1942).*

| | No. of Cases | | No. of Days Lost | Amount of Indemnity Paid | Cost of Medical Care | Total Costs |
|---|---|---|---|---|---|---|
| | Temporary Disability | Permanent Disability | | | | |
| 1941: | | | | | | |
| Dermatitis | 359 | | 6,686 | $17,153 | $12,114 | $29,267 |
| Plant poisoning | 182 | | 2,123 | 4,078 | 3,450 | 7,528 |
| Totals | 541 | | 8,809 | $21,231 | $15,564 | $36,795 |
| 1942: | | | | | | |
| Dermatitis | 560 | 1 | 8,863 | $22,628 | $16,655 | $39,283 |
| Plant poisoning | 134 | | 1,602 | 3,754 | 2,596 | 6,350 |
| Totals | 694 | 1 | 10,465 | $26,382 | $19,251 | $45,633 |

TABLE 2. *Average Number of Days Lost and Average Indemnity and Cost of Medical Care in Cases of Dermatoses in Wisconsin (1937–1942).*

| Data | 1937 | 1938 | 1939 | 1940 | 1941 | 1942 |
|---|---|---|---|---|---|---|
| Average days lost | 23 | 24 | 23 | 19 | 19 | 16 |
| Average indemnity | $53.00 | $60.00 | $61.00 | $48.00 | $48.00 | $40.00 |
| Average cost of medical care | $37.00 | $37.00 | $46.00 | $35.00 | $35.00 | $31.00 |

tion paid and medical expenses between January 1, 1940, and October 1, 1944. The total expense to the company was $94,578.00. The average amount of compensation paid was $76.00 and the average cost of medical expense was $49.00, making the average expense per case $125.00. If the cases in which no compensation was paid are deducted, the average compensation paid per case rises to $94.00 and the average medical expense to $52.00. The increase in the number of cases this year in the ten months of the year is especially striking, but there is no information concerning the large increase in the number of cases in which no compensation was paid. There were only 6 cases in which the total expense was over $1000.00, the largest amounting to $3515. No data concerning duration of disability or wages lost were available.

Table 3. *Compensation for Cases of Occupational Dermatoses in New York State (1943).*

| Type of Case | No. of Cases | Duration of Compensation | | Compensation Awarded | |
|---|---|---|---|---|---|
| | | Total wk. | Average wk. | Total | Average |
| Fatal | 1 | 1,000 | 1,000.0 | $10,403.00 | $10,403.00 |
| Partial disability | 47 | 1,336 | 28.4 | 22,856.00 | 486.00 |
| Temporary disability | 1,652 | 12,269 | 7.4 | 189,052.00 | 114.00 |
| Totals | 1,700 | 14,605 | | $222,311.00 | |
| Averages | | | 8.6 | | $130.77 |

Table 4. *Data in 753 Cases of Dermatitis (1940–1944).*

| Year | No. of Cases | Compensation Paid | Cases with No Compensation | Medical Expenses Paid | Cases with Medical Expense | | |
|---|---|---|---|---|---|---|---|
| | | | | | None | over $500.00 | over $1,000.00 |
| 1940 | 20* | $1,705 | 5 | $1,988 | 2 | 0 | 0 |
| 1941 | 65 | 9,726 | 9 | 3,947 | 6 | 6 | 2 |
| 1942 | 176 | 10,795 | 27 | 8,575 | 7 | 3 | 1 |
| 1943 | 174 | 13,526 | 27 | 8,954 | 12 | 7 | 2 |
| 1944 | 318* | 21,720 | 75 | 13,642 | 21 | 6 | 1 |
| Totals | 753 | $57,472 | 143 | $37,106 | 48 | 22 | 6 |

*Incomplete figure.

Undoubtedly further effort would reveal other figures of similar nature. Other states were approached as well as other insurance companies, but the data were not readily available. There are thus very few figures indicating the medical expenses and compensation paid for these cases. There are even fewer data on the duration of disability and the amount of wages lost. There is only an occasional reference to the end results, that is, the ability to return to the same job or the need for transfer to some other job.

These extremely incomplete figures on duration of disability, wages lost, medical expenses and compensation paid for occupational dermatoses are impressive enough to warrant more attention by manufacturers, insurance companies and state accident commissions. Further studies should be made in an effort to determine the actual losses over a wider area and in a larger series of cases. The size of such losses should stimulate further efforts toward earlier detection and diagnosis and, a search into the factors prolonging disability, should eventually lead to measures that will prevent or lessen such losses.

Much has been written concerning the causes of disability and concerning hazardous occupations so far as occupational cutaneous disease is concerned, but it is not generally realized in what a high percentage of the cutaneous cases seen by the average physician a causal occupational factor arises. If all persons, including housewives, using soap and water in their work were considered, an occupational factor would need to be reviewed in 15 to 20 per cent of all dermatologic cases. It is therefore important for the physician to have in mind the criteria by which he is to determine the correct occupational liability. The National Committee on Occupational Dermatoses after much discussion made a report on this subject, which was published in 1942.[3] The use of the criteria given in this report would be a distinct aid in the recognition of occupational cases, at least from the medical standpoint. In making decisions regarding the liability for disability from occupational dermatoses, the physician should answer the following questions, which are based on these criteria.

Has the patient been in contact with an agent known to have produced similar changes in the skin?

Is the time between exposure to the agent claimed and the onset of disease correct for that particular agent and for the particular disease that the patient presents?

Is the place on the body where the disease began the site where the greatest exposure has occurred?

Is the site of the greatest skin involvement the place where the greatest exposure has occurred?

Are the lesions present consistent with those known to have followed a similar exposure or trauma?
Is the person employed in an occupation in which similar cases have occurred?
Do any of the other workers at the same job have similar manifestations on the skin due to the same cause?
If the diagnosis is one of dermatitis, have attacks occurred after exposure to the agent claimed, and has relief occurred after cessation of exposure?
If the diagnosis is one of dermatitis, and skin tests have been done by a generally accepted technic and interpreted by a competent physician, are the results consistent with the facts obtained from the history and physical examination?
Is the diagnosis that of a disease in which a causal factor due to occupation has been established beyond reasonable doubt?
Has the patient been exposed outside his work to an agent that might have caused a similar eruption?
Is there any doubt concerning the diagnosis?

The two most influential factors that increase the difficulty of making a diagnosis and of determining the liability of occupational dermatoses are the length of time that has elapsed from the onset until examination and the failure to obtain an accurate history of the early period of the eruption. If the cases can be examined early and the satisfactory fulfillment of the criteria obtained, a definite diagnosis of occupational liability can be made. If the essential items of the criteria do not apply fully, all that can be said of a particular case is that it is *probably* one of occupational dermatosis. In numerous cases the only statement that can be made is that the case is of *possible* occupational origin; in other words, a reasonable or more than reasonable doubt exists of the industrial causation. For example, if the answers to the first ten questions are in the affirmative, the skin disturbance is undoubtedly occupational. If the answers to the last two questions are in the affirmative, there is reasonable doubt that occupational liability exists.

The most frequent diagnosis in patients incurring disability or receiving compensation for cutaneous disturbances is dermatitis venenata or contact dermatitis, but there are various other diagnoses in which consideration must be given to the occupational liability. As I[4] have previously stated, the most frequent diseases in which an occupational factor can occur are as follows:

| NONINFECTIOUS DISEASES | INFECTIOUS DISEASES |
|---|---|
| Acne | Actinomycosis |
| Burns | Anthrax |
| Calluses | Blastomycosis |
| Chilblains | Erysipeloid |
| Dermatitis, acute or chronic | Impetigo |
| Eczema | Paronychia |
| Epithelioma | Pus infections |
| Folliculitis | Syphilis |
| Keloids | Tinea |
| Keratoses | Tuberculosis |
| Radiodermatitis | |
| Ulcers | |

There are many other diseases in which an occupational factor may be a cause. It is to be remembered that in some states the complications of occupational disease and the effect of pre-existing disease in enhancing disability are also compensable. This broadens the scope of the compensation laws, especially when all the possible complications of therapy are considered. The record of a diagnosis should include the fact that the dermatosis is occupational and should state the type of disease present (dermatitis, keloid and so forth), the cause (if known) and the occupation. With these data cross-filed, a study of various series of cases at a later period with reference to disability and compensation is an easy matter.

The type of disability that results from cutaneous disease is fully as varied as that in disease of any other organ. Most patients suffer a temporary disability, and many suffer a total disability for a shorter or longer period. A few are permanently disabled, in that they are unable to return to their jobs or must give up working altogether.

An occasional death occurs as the result of some complicating factor. Some patients have a partial disability in that they are slowed up in their work or can do only part of it or are limited to part-time work. An occasional permanent partial disability occurs.

The duration of the disability has many contributing factors. The most hazardous agents and the occupations in which the majority of cases occur have been discussed in numerous reports, and much preventive work for such occupations has already been outlined.[5] Carelessness on the part of the worker or his failure to observe the preventive regulations concerning a particular product are often fundamental causes of a long disability, and in many cases deliberate failure to report an eruption for fear of losing time or for some other reason has caused a longer disability than would otherwise have resulted. Failure to detect cases early, whether on the part of the foreman, the nurse or the physician, is also a contributing factor. Additional exposure after the onset of irritation and the late commencement of treatment are extremely important in the prolongation of disability. Failure to have troublesome cases examined early by a physician with some training in cutaneous medicine is responsible for many cases of excessive disability. In a series of 40 shoeworkers studied, it was found that the average period before examination by a dermatologist was five weeks. The longest period was two years, and several cases were allowed to go for four to six months before having the possible benefit of more experienced advice.

It is also necessary to be sure that the worker is not suffering from a nonoccupational disease, such as erythema multiforme or lichen planus. Self-medication, usually of the wrong type, often contributes to extra disability. The use of an agent too strong for the skin adds its share to the toll. Sensitization of the patient, either to substances that he contacts during work or to agents used in therapy, occurs in a far larger proportion of occupational cases than is generally believed and may

add materially to the period of disability. Finally, secondary infection of a wound or a dermatitis may increase the time lost from work.

The cases with long disability that have been mentioned, as well as the two deaths mentioned by the New York report, are unusual, but they emphasize the possibility of long-continued disability and of high costs of an occupational dermatosis. Although only a limited number of figures regarding time and money lost from disability due to occupational dermatoses are available, these suggest that a further study should be made to ascertain the actual amount of loss incurred from this type of disease. Many dermatologists believe that in occupational dermatoses the disability can be reduced and in many cases prevented, and that under normal working conditions much can be done to lessen the toll that these skin disturbances take. Industry is interested in the prevention of such cases and the avoidance of complications, or at least in limiting the time lost to a short period of disability, with the desire to return workers early and to provide for the full production of the product involved. The employee, on his side, desires to maintain or even improve his earning power and is anxious to avoid lost time from illness. The medical profession is interested in working with both parties, detecting these cases early and providing treatment so that the period of disability will be little or none.

For the physician who sees many industrial cases — for example, the medical director of a large or small industrial medical department — there are certain measures that will aid him in preventing lost time and in reducing compensation paid and other expenses. He can find his cases early by training foremen and nurses to detect skin manifestations, and can send these workers to the plant dispensary for early therapy. If he is able to diagnose cases early or to obtain adequate consultation, a good beginning will have been made. If a cutaneous condition is found, it is necessary to exclude the causative factors, if humanly possible, by obtaining aid in devising adequate preventive measures. Early treatment should be soothing, and care should be taken to avoid secondary infection. The selection of proper employees for hazardous jobs and a follow-up of those returning to work after a dermatitis will aid in preventing later recurrences and more disability.

* * *

In the larger aspects of losses due to occupational disability caused by cutaneous manifestations, there are several accomplishments to be attained. There should be further basic studies concerning the physiologic and chemical activities of the skin. Factories and working places should be provided with means and personnel for the early detection and treatment of occupational dermatoses. There should be available more statistics and more end-result studies of such occupational cases. There should be capable dermatologic representation on boards and commissions dealing with occupational dermatoses. There should be improved instruction in occupational dermatoses and in the dermatologic aspects of legal medicine pertaining to compensation cases. There should be adequate legislation in every state to cover all types of occupationally acquired dermatoses. Lastly, the medical profession must work with the law in the most effective and impartial analysis of claims and in the just disposition of cases.

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# THE DEVELOPMENT OF ANATOMICAL LAWS IN THE STATES OF NEW ENGLAND

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RECORDS of human dissection in classical Greece and Rome are scanty. Traditions exist that some physicians of the era of Hippocrates, among them Democritus, dissected the human body, but little accurate knowledge of the structure and arrangement of the internal human organs is found in the writings before the Christian Era. The details of human anatomy were inferred from dissection of domestic animals, some differing widely from man in structure. Galen is reported to have dissected monkeys, which are primates, resembling man in structure, that were household pets in Rome in his time.

The most considerable addition to the knowledge of human anatomy made before the Christian era occurred at the Akademie of Alexandria — often called the University of Alexandria — in the third century before Christ. Ptolemy I decreed that the bodies of criminals who were executed and those of prisoners who died in Alexandria should be surrendered to the physicians for the study of anatomy. This privilege is the first recorded legal provision for human dissection; it continued for several years. Herophilus, Erasistratus and their followers discovered many new facts in human anatomy and introduced new terms of nomenclature, some of which are in use at the present time.

Little was added to the knowledge of human anatomy in the first millennium of the Christian era. Popular prejudice against human dissection brought severe penalities to those who attempted it, a prejudice in part the result of sentiment but also involving factors of religious belief concerning the effect of dismemberment of the body on the happiness of the soul that had departed from it. Opinion is not in agreement concerning the early attitude of the Catholic Church toward dissection. Some ecclesiastical edicts are interpreted as a ban on any dismemberment of the dead body or incisions of it. Emperor Charles V, the patron of Vesalius, in 1556 asked the theologians of the University of Salamanca whether it was a violation of conscience and a suspicion of criminality to cut up dead human bodies "*et repondisse Universitatem, licere.*"

The establishment of medical schools and universities in Europe in the Middle Ages brought institutional teaching of medicine, including anatomy. Reigning monarchs and princes on the continent of Europe occasionally decreed that the body of an executed felon should be delivered to a physician or surgeon for dissection and public demonstration. The recorded instances are few. Important features of this phase of legal provision for human dissection include not only the rarity of its occurrence but also the fact that it depended entirely on the favor or caprice of the individual ruler.

Occasional dissections occurred in Italy in the thirteenth century. Mondino did them early in the fourteenth century, as shown in his textbook on anatomy. Human dissection is first recorded under the Faculty of Medicine in Paris in the fifteenth century, and late in the sixteenth century a statute in that city directed that at least two public dissections be done in each year. The work of Vesalius came in the sixteenth century.

## EARLY PROVISIONS IN GREAT BRITAIN

Inasmuch as American anatomical laws can be traced back to earlier procedures in Great Britain, further mention of conditions in Europe are restricted to events in England and Scotland.

The first medical-practice act in England was passed in 1421 in the reign of Henry V.[1] The first enactment in the American colonies that can be called a medical-practice act was passed by the Virginia Assembly in 1638–1639 and was re-enacted in 1645–1646. It was mainly concerned with professional fees. The statute was amended and extended in 1657–1658.[2, 3] The first similar act in New England was passed in the Massachusetts Bay Colony in 1649.[4] It concerned skills and the limits of what men practicing medicine should attempt. Anatomy is mentioned in none of these laws.

The magistrates of Edinburgh in 1505 granted a "seal of cause" or charter to the Guild of Surgeons and Barbers of that city, which was approved by James IV early in 1506. Among its many provisions was one granting annually the body of an executed criminal for dissection.[5]

The first reference to such a chartered right in the acts of the English Parliament was the enactment of a charter for the Guild of Barbers and Surgeons of London in 1540 in the reign of Henry VIII.[6] One of the provisions of this charter merits quotation.

> That the said masters or governors of the mystery and commalty of barbers and surgeons of London and their successors yearly forever . . . shall and may have and take without contradiction four persons condemned, adjudged and put to death for felony . . . for anatomies without any further suit or labour to be made to the King's highness, his heirs or successors for the same; and to make incisions of said dead bodies . . . for further and better knowledge, instruction, insight, learning, and experience in the science and faculty of surgery.

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This act was the beginning of statutory provision for human dissection on which is based the present anatomical laws in the United States. It was not until three hundred and fifty years after its granting, however, that adequate laws were enacted in New England.

Queen Elizabeth in 1564 granted a special "charter for anatomies" to the College of Physicians in London. This provided that four bodies of executed felons be annually delivered to that organization for anatomizing.[7]

Charles II in 1663 increased the annual number to six "for further and better knowledge, instruction, insight, learning, and experience in the faculty and science of physick and chirurgery," repeating the phraseology in the charter granted to the Guild of Barbers and Surgeons by Henry VIII. He directed in addition that after these bodies had been anatomized the remains should be "decently buried."[8]

The body of English law under which the American colonies were governed had two distinct parts, namely, statutory law and common law. James Kent,[9] eminent early American writer on law, defined the common law as follows:

> The common law includes those principles, usages and rules of action applicable to the government and security of person and property, which do not rest for their authority upon any express and positive declaration of the will of the legislature.

The common law developed by accumulation of precedents such as an act of a magistrate or a judge to which no objection was made by higher authority. Obviously, it is difficult to determine the exact date of the first action that by repetition became a common law. Even the approximate date of the beginning of another phase of legal provision for human dissection is unknown. Apparently it was prior to the enactment of the charter of 1540, the first English statutory provision. This procedure was firmly incorporated in English common law before the first settlement in New England in 1620.

Under the common law a judge was permitted in sentencing a murderer to execution, to add the further penalty that his body be dissected; this was considered an additional disgrace. Blackstone[10] wrote in his commentaries regarding this permission to judges: "In very atrocious crime other circumstances of terror, pain and disgrace were superadded . . . such as in murder, public dissection."

The traditional British attitude toward the sanctity of habitation placed arson and burglary next in degree of atrocity to murder; both were capital crimes. No reference, however, in either English statutory or common law, can be found to the imposing of the additional penalty of dissection on those sentenced to death for either arson or burglary. This disgrace of dissection appears to have been reserved for those convicted of murder, considered the most atrocious crime next to high treason.

This procedure under common law became English statutory law in 1752 in the reign of George II, when Parliament passed an act containing the following provision:

> The Judge before whom any person is found guilty of wilful murder shall pronounce sentence immediately after conviction unless he sees cause to postpone it; and shall in passing sentence direct him to be executed on the next day but one (unless the same shall be Sunday, and then on the Monday following) and that his body be delivered to the surgeons to be dissected and anatomized; and that the Judge may direct that his body be hung in chains but in no wise to be buried without dissection. . . . But a power is allowed to the Judge upon good and sufficient cause, to respite the execution and relax the other restraints of the act.

The body of this statute made the further penalty of dissection mandatory, but the final sentence of the section quoted modifies it to become only permissive. The title of this statute is "An Act for better preventing the horrid Crime of Murder."[11] The preamble begins as follows:

> Whereas the horrid crime of murder has been more frequently perpetrated than formerly . . . and whereas it is thereby become necessary that some further terror and peculiar infamy be added to the punishment of death. . . .

These words show conclusively that dissection was considered "a peculiar mark of infamy."

The statute provided that if the execution occurred in the City of London or in the County of Middlesex, the body of the murderer should be conveyed immediately to the hall of the Surgeons' Company to be dissected and anatomized. Further penalties were provided for any attempt to prevent dissection. If the execution occurred outside the County of Middlesex, the dissection was to be done if some surgeon could be found who desired to do it; otherwise the body was to be buried.

## Early Anatomical Laws in New England

### *The First Phase*

The common-law permission to a judge to add the further penalty of dissection to a sentence of execution for murder was familiar in the early years of the settlement of New England. *The Body of Liberties*[12] was adopted in the Massachusetts Bay Colony in 1641 as the first code of laws. One rule in this reads as follows:

> No man condemned to dye shall be put to death within fowere dayes after his condemnation unless the Corte see spetial cause to the contrary as in the case of martiall law, nor shall the body of any man so put to death be unburied twelve hours unless it be in the case of Anatomie.

This rule shows that the bodies of executed murderers were available for dissection in New England as early as twenty years after the first landing at Plymouth. The word "anatomizing" and the phrase "making an anatomie" were commonly used in England and the American colonies to designate the procedure of dissection.

The first record of a human dissection in New England occurs in a communication written by a

colonial author and published in London in 1647.[13] It reads as follows:

> Our young students in Physic may be trained up better than yet they bee, who have onely theoretical knowledge and are forced to fall to practice before ever thay saw an anatomie made, or duly trained up in making experiments, for we never had but one anatomie in the country, which Mr. Giles Firmin (now in England) did make and read upon very well, but no more of that now.

The date and place of this dissection are not recorded. Giles Firmin, a physician-clergyman, was in the Massachusetts Bay Colony from 1632 to 1646, at first resident in Boston but from 1638 to 1644 resident at Ipswich. It is probable that the dissection was made at Ipswich. One writer suggests that it was made on a body secured by robbing a grave. Likelier it was on the body of an executed murderer, a procedure permissible under the common law and the code of 1641.

The rarity of provision of the bodies of executed murderers for dissection in the early colonial period is indicated by a resolution adopted October 27, 1647, by the Governor and Council of the Massachusetts Bay Colony,[14] reading as follows:

> We conceive it very necessary yt such as studies physick and chirurgery may have liberty to read & to anatomize once in four years some malefactor in case there be such as the Courte shall alow of.

Records of human dissection in colonial New England are rare. There were no medical colleges and no medical journals, and such events would not be likely to be published in any of the few newspapers of the eighteenth century because of the public aversion to the procedure.

Dr. William Hunter delivered lectures on anatomy at Newport, Rhode Island, in 1754, 1755 and 1756.[15] These were attended by both physicians and laymen. His advertisements announcing the lectures made no mention of demonstrations of a dissected human cadaver. Some writers have considered this omission as evidence that no demonstrations were given, but Dr. Hunter would probably not have mentioned such an intention in an advertisement in a newspaper, even if he could have foreseen that some favoring judge would provide the body of an executed murderer.

An earlier record is found outside New England. Dr. Peter Middleton and Dr. John Bard made a human dissection in New York City in 1750.[16,17] The body was that of a murderer executed in New Jersey. Dr. Thomas Cadwalader, probably before 1751, and Dr. William Shippen, beginning in 1762, gave lectures on anatomy in Philadelphia in which they demonstrated dissected human bodies.[18]

These few records indicate the probable rarity of dissection of bodies legally acquired in the colonial period in New England. The only legal source was the body of an executed murderer. One writer[19,20] says that the bodies of suicides were available for dissection in the colonial period. A law adopted in the Massachusetts Bay Colony in 1660[21] states that a suicide is denied "the privilege of being buried in the common burying place of christians, but shall be buried in some common highway . . . and a cart load of stones laid upon the grave as a brand of infamy. . . ." A search of the colonial laws reveals no provision for dissection of the body of a suicide, nor did the English common or statutory law carry such a provision, although the bodies of suicides were available at Edinburgh shortly before 1700 under an act of the local magistrates.

Dr. Harrington,[22] writing about 1905, states that it was the rule in Massachusetts at the close of the Revolutionary War for the court to have discretion in allowing the dissection of the bodies of those executed for murder, arson or burglary. I am unable to confirm this statement so far as it relates to the bodies of those executed for arson or burglary. This statement is made in connection with several others that are apparently taken from an article published in 1896.[23] Dr. Harrington cites this article. A careful reading of it brings the conclusion that he misinterpreted a somewhat obscure passage. Neither the English common law nor statutory law, as already stated, provided that the bodies of those executed for arson or for burglary might be directed to be dissected, as were the bodies of murderers. No provision for dissection of the bodies of those executed for arson or burglary occurs in the acts of the Legislature of Massachusetts in the colonial period nor in the early national period. Such a statutory provision was made in New York in 1789. It therefore appears that the only legal provision for human dissection in the colonial period in New England came from an occasional additional sentence by a judge, in condemning a murderer, that his body should be dissected.

An additional source was provided in Massachusetts in 1784. All the colonies enacted laws against dueling in the eighteenth century. The law was revised in Massachusetts in 1784 when a statute entitled "An Act against Duelling" was passed.[24] One of the provisions of this act provided that the body of one killed in a duel might either be buried without a coffin in a public place with a stake driven through the body — an ignominious form of burial — or be given to a surgeon for dissection. In the case of one convicted of "murder in a duel" and executed, however, the body must be given to a surgeon for dissection. If no surgeon applied for the body, the ignominious form of burial was to be used. This statute is the first instance in New England of a law that was mandatory in regard to the giving of a dead body for dissection. A statute was enacted in 1805 in Massachusetts[25] in which dueling was included with murder and some other crimes. The mandatory feature regarding the body of a duelist was continued. The specific law of 1784 regarding dueling was repealed in 1806, because the penalty for the crime was covered by the statute of 1805.[26]

In none of the laws of the several states of New England except that of Massachusetts was the feature of the dissection of the body of one engaged in a fatal duel included until the District of Maine became the State of Maine in 1820. Its first legislature adopted verbatim many of the laws of Massachusetts under which it had been governed as the District of Maine. One of these[27] provided for the dissection of the bodies of those who had engaged in a fatal duel. Fatal duels were few, so that these laws in Massachusetts and Maine provided little additional legal material for dissection.

The provisions for the dissection of the bodies of murderers and duelists were not specifically designed to advance the study of anatomy. They were in every case a further penalty for crime, and the availability of the body for dissection was only incidental to the main purpose of such provisions. Therefore these laws were not strictly anatomical laws, although they did incidentally provide legal material for the study of anatomy.

The first legislative act in the northeastern part of the United States that shows by its wording a purpose to provide for the study of practical anatomy was the direct result of a public disturbance. It became rumored in New York City in May, 1788, that a dissection was in progress on a body illegally disinterred. The public clamor resulted in a riot that became so serious that the state militia was called out to disperse the mob. This episode was known as "the doctor's mob."[28]

The Legislature of New York at its next session enacted on January 6, 1789, a statute entitled "An Act to prevent the Odious Practice of digging up and removing for the Purpose of Dissection, dead Bodies interred in Cemeteries or Burial Places."[29] The first section provided penalties for the offense; the second is important enough to be quoted.

> In order that Science may not in the Respect be injured by preventing the Dissection of proper Subjects, be it further enacted by the Authority aforesaid, that the Justices of the Supreme Court or of any Court of Oyer and Terminer and Gaol Delivery, in this State, from Time to Time, when any Offender shall be convicted before them or any of them of Murder, Arson, or Burglary for which he or she shall be sentenced to suffer Death, may at their discretion add to the Judgement that the Body of such Offender shall be delivered to a Surgeon for Dissection; and the Sheriff who is to cause such Sentence to be executed shall accordingly deliver the Body of such Offender, after Execution [is] done, to such Surgeon as such Court shall direct for the Purpose of aforesaid; Provided always that such Surgeon, or some other Person by him appointed for the Purpose, shall attend to receive and take away the dead body, at the Time of Execution of such Offender.

This law was not copied in any state in New England, but the fact that it put those executed for arson and burglary in the same class as murderers may have been an influence in laws later enacted in states of New England that extended the privilege of dissection to the bodies of other than murderers, especially in the law of 1824 in Connecticut, to be mentioned later.

The several states of New England, with the beginning of the national period, enacted codes of laws, but except in so far as these were contradictory to the English common and statutory law, under which these states had existed as colonies, the body of English law continued in force. James Kent[30] wrote in his commentaries on American law of the early national period as follows:

> But though the great body of common law consists of a collection of principles, to be found in the opinions of the sages or deducted from universal usage and receiving progressively the sanction of the courts, it is nevertheless true that the common law, as far as it is applicable to our situation and government, has been recognized and adopted, as one entire system in the constitutions of Massachusetts, New York, New Jersey and Maryland. It has been assumed by the courts of justice, or declared by statute, with like modifications, as the law of the land in every state.

Therefore the availability of the bodies of executed murderers for dissection at the discretion of the sentencing judge continued to be a source of material for anatomical study. In some states the procedure was expressly stated in the statutes; in others it was followed only under the ancient common law. In one state of New England this procedure is contained in the anatomical law now current.

Thus the availability for dissection of the bodies of those executed for murder, and after 1784, of those engaged in duels in Massachusetts, was the only legal source of material for dissection in any state in New England during more than two hundred years after the first settlement in 1620. Few bodies were available from this source. A statistical compilation made in Massachusetts in 1830 showed that for the thirty years from 1800 to 1829, inclusive, the total number of executions in that state averaged only little over two a year, and by no means all those to be executed were further sentenced to be dissected.

Inasmuch as this provision was only incidental to the punishment of arch criminals, in the public mind dissection was placed in close relation to major crime and brought the conviction that dissection was an ignominy, and that anyone whose body was dissected suffered posthumous disgrace and was thereby allocated to the same class as murderers, a belief that accentuated the aversion to dissection arising from sentiment.

The number of medical colleges increased until in 1823 there were eight of them in New England. There was a corresponding increase in the number of medical students. All students were expected to do human dissection, although no such requirement was printed in the announcements or catalogues of the colleges. The provision for legal acquisition of bodies for dissection was no broader than in 1784, forty years earlier, when there was only one medical college in New England. The only method of adequate provision for human dissection was increasing recourse to grave robbing. The alternative was the abandonment of the teaching of practical anatomy.

Elsewhere I[31] have discussed the extent of grave robbing in New England.

A slight relief came in Connecticut in 1824 with the enactment of a law providing that all bodies of those who died in the State Prison or who were executed for any crime, unless claimed by kindred, should be delivered for use in the teaching of anatomy.[32] Previously the state had maintained a cemetery at the State Prison in which were buried the unclaimed bodies of those who died or were executed in that institution. This was the first law in New England to provide for the anatomical study of bodies other than those of murderers and duelists.

*The Second Phase*

The extension of legal sources of material for human dissection in the states of New England was the indirect result of a series of events in another country, events familiar to all those interested in medical history. The immediate cause of these events is less well known.

Edinburgh was a center of medical education in the early nineteenth century. Anatomy had been taught in institutions in that city for more than a hundred years — since early in the eighteenth century. The methods of visual instruction, however, were restricted to charts and demonstrations of prosected cadavers in amphitheaters. Only a few favored assistants of the professors had the privilege of personally engaging in dissection. The great majority of medical students never applied a scalpel to a dead human body and got no nearer to a cadaver than a seat in an amphitheater during a demonstration. These methods of teaching required only a few cadavers, those bodies of prisoners dying in the House of Correction.

A change came in 1826 when the College of Surgeons of Edinburgh announced that thereafter every medical student who desired the degree of Doctor of Medicine must personally engage in dissection. The University of Edinburgh made a similar requirement a year later.[33] This greatly increased the demand for dead bodies. The prices paid for them advanced, causing increasing activity of the resurrectionists that led to publicity and to public clamor in Scotland and England regarding grave robbing. The British newspapers published much on the subject, and a considerable part of this material was reprinted in American newspapers. The knowledge of events in Great Britain reached legislators as well as all members of the American public, and it seemed to the medical profession a favorable time to secure legislative reform of the inadequate legal provisions for the study of practical anatomy.

The public became conscious that grave robbing was more frequent than had been suspected, and the prejudices against dissection and medical colleges were accentuated. The medical profession had long desired legal provision of material for teaching practical anatomy, for grave robbing was distasteful to all its members. Individual physicians and small groups of them had repeatedly asked legislatures to provide relief. This was usually spoken of as "legalizing anatomy," a somewhat misleading phrase, because anatomy was never illegal in New England, nor was dissection. The illegality pertained only to some methods of procuring dead bodies.

Legislators had given little heed to these requests of physicians because of the well-known popular aversion to dissection. Occasionally a bill was introduced in a legislature by a physician who was a member of the body. Such bills rarely got beyond reference to a committee that declined to report the bill for a vote.

The first concerted action in New England began in the Massachusetts Medical Society on February 4, 1829, when Dr. Abel Lawrence Peirson introduced a resolution to appoint a committee of the society to seek legislation.[34] He was appointed chairman of a committee and continued as chairman of an enlarged committee appointed a few months later. He gave much time and effective effort to the work. A survey of the activities of the next few years leads to the conclusion that Dr. Peirson more than any other one man deserves the credit of securing the legislation that followed. This conclusion is not in accord with that of another writer,[35] who in 1896 stated that Dr. John C. Warren, professor of anatomy in the Harvard Medical School, was largely responsible for the legislation. This writer was a grandson of Dr. Warren.

A long, argumentative circular letter was sent to each member of the medical society asking him to try to influence such members of the Legislature as he knew personally.[36] This letter was dated September 1, 1829, at Salem, where Dr. Peirson was in practice, and was probably written by him. A bill was introduced in the session of the Legislature of January, 1830. It was referred to a committee, which reported that it "did not think it expedient to propose any alteration of the laws at the present time; because in a community like ours, it is necessary that laws should proceed from and be supported by public opinion." [37]

The committee of the Medical Society sought to educate the public, and articles signed by members of the committee were published in newspapers. A pamphlet was prepared arguing the desirability of the proposed law, and 10,000 copies were ordered printed for distribution throughout the state.[38]

The bill was again introduced at the session of May, 1830. The Governor, in his address at the opening of the Legislature, discussed the desirability of better provision for teaching anatomy.[39] This part of his address was referred to a special committee. The bill was postponed to the next session.

An argument publicly advanced was that adequate legal provision for human dissection would

benefit the poor, because if all medical students could have instruction in practical anatomy all physicians could acquire this knowledge instead of its being largely restricted to those who could go to Paris, where material for dissection was abundant, and who on returning charged larger fees and were usually employed only by the well-to-do.[40]

On invitation, Dr. John C. Warren addressed a joint session of the Legislature on February 2, 1831, on the subject.[41] He was a member of the committee of the Massachusetts Medical Society and professor of anatomy and surgery in the Harvard Medical School, and was well and favorably known throughout the state. The bill was passed in the session of January, 1831, under the title, "An Act more effectively to Protect the Sepulchres of the Dead and to Legalize the Study of Anatomy in Certain Cases."[42] This is considered the first effective anatomical law in any English-speaking country. It antedated the Anatomy Act of the English Parliament by fifteen months.[43]

The new Massachusetts law permitted civil officials to surrender for dissection any body that must otherwise be buried at public expense, with some exceptions, thus going far beyond the former limitation to the bodies of major criminals. Preference in delivery of such bodies to physicians was to be given to those who were teachers in a legally chartered medical college in the state. Harvard Medical School and the Berkshire Medical Institution were the two chartered medical colleges in Massachusetts at that time. The law received public approval.[44]

This legislation was defective in two major respects, the first being that it was not mandatory, but only granted permission to civil officials to deliver bodies for dissection at their discretion, and the second being that it included several exceptions that tended to nullify its purpose. Officials were not permitted to deliver for dissection the body of any stranger, any town pauper, any person who during life had requested that his body be buried or any person whatsoever if within thirty-six hours a kinsman or friend requested that the body be buried.

The earlier sections of the law provided penalties for disturbing graves or disinterring bodies and by specific statement repealed former laws on this subject. This repeal was one of the most important features of the new law, because the law of 1815, in Massachusetts, had provided the same penalties for possession of a disinterred body as for the disinterment itself.[45] Possession was defined as presence of a body on premises controlled by the accused, and the penalties were heavy fines and imprisonment. The matter of possession was not mentioned in the law of 1831. Although the primary stimulus of the activities that secured this law was a series of events in Scotland and the publicity given to them, I[46] have elsewhere expressed the opinion that local events in Massachusetts a few years earlier had a large influence, especially in securing the effective interest of Dr. Peirson.

The law was somewhat improved by amendments in 1834.[47] During the next forty years the law was amended several times without extensive modification of its provisions. The amendments concerned additions to the list of public institutions from which bodies might be delivered and changes in the titles of governing authorities of these institutions.

The enactment of this law in Massachusetts had an effect in neighboring states. Connecticut passed a law in 1833 providing that all bodies to be buried at public expense were to be available for dissection,[48] but this was repealed exactly a year later.[49]

An anatomical law was passed in New Hampshire in 1834 that was a close copy of that of Massachusetts,[50] but this was repealed in 1842 after having been in force for only eight years.[51] A group of physicians in Vermont in 1835 tried unsuccessfully to secure passage of a law similar to those of Massachusetts and New Hampshire. I have found no record of attempts at this time to pass similar laws in Maine or Rhode Island.

To summarize the events of the second phase, an anatomical law was passed in Massachusetts in 1831 and was continued on the statute books. A law of this nature was passed in Connecticut in 1833 and was repealed after one year, although the law of 1824 in that state regarding the bodies of those who died or were executed in the State Prison continued in force. A law passed in New Hampshire in 1834 was repealed after eight years. No similar law was enacted in Maine, Rhode Island or Vermont.

The fact that Massachusetts secured and maintained an anatomical law, whereas in Connecticut and New Hampshire such laws were passed but soon repealed and in the three other states of New England no such laws were secured, suggests some influences in Massachusetts not found in the other states. Two may be conjectured, the first being that the extensive program of education of the public concerning the desirability of an anatomical law in Massachusetts made by the Massachusetts Medical Society in 1830 had a continuing effect. The other is that many members of the Legislature in Massachusetts were graduates of Harvard University, which may have had some influence on these legislators regarding a law that much concerned the medical school of that university. New York was the only other state in which an anatomical law enacted before 1860 continued in force.

*The Third Phase*

The influences that brought about a third phase of anatomical laws in New England were not local to that area. The organization of the American Medical Association in 1847 had two major objects. One of these was the improvement of medical educa-

tion. In that society and in many state medical societies papers were read, committees appointed and resolutions adopted, all of which had no marked immediate result. In a few colleges the length of the medical session was slightly increased, but the leading deficiencies of lack of standards in preceptorial teaching and the repetitive curriculum were not eliminated, and the medical-education program of 1860 was not materially better than that of 1830.

Some concerted demonstration of educational weakness was needed to bring reform. The first establishment of medical colleges in the American colonies was the result of evidence of medical deficiencies in the French and Indian War.

Similarly, it was the Civil War that influenced improvement needed in that era. Medicine and surgery had shown many deficiencies in the field. After the close of the war a definite and concerted action began to improve the program of medical education. A convention of medical teachers held in 1867 advocated lengthening of the teaching session, increase of the number of sessions to be attended before receiving the degree of Doctor of Medicine and a graded curriculum. Emphasis was placed on needed improvement in certain subjects. Two of these were practical anatomy and operative surgery on the cadaver. Such extensions would require the availability of more cadavers.

These conditions were probably responsible for introducing the events of the third phase of anatomical laws, which was the extension to other states in New England of laws similar to the one that had been in force during more than thirty-five years in Massachusetts.

The first legislative response in New England occurred in March, 1869, with the passing of a law in Maine permitting bodies that must otherwise be buried at public expense to be surrendered for dissection.[52] A similar law was passed three months later in New Hampshire, which closely resembled the law that had been in force in that state from 1834 to 1842.[53] Connecticut delayed until 1871.[54] Vermont did not join these three states in passing a permissive law at this time. Rhode Island has had no medical college since 1828 and has never had an anatomical law.

The anatomical laws passed in Maine, New Hampshire and Connecticut from 1869 to 1871, together with the law of Massachusetts passed in 1831, had certain basic similarities, but some of them had provisions not found in other states. The common provisions were three: first, that a body delivered for dissection should be used only for the advance of anatomical and surgical science; second, that the body should not be transported beyond the boundaries of the state in which it was surrendered; and third, that after the body had been used for dissection the remains should be given decent burial.

The Maine law had a peculiar provision as its first section, which was that if a person requested or consented before death that his body be used to advance anatomical science, it might be so used if no kindred objected. This is in direct conflict with the legal principle in force for many centuries that a person cannot devise by will or by contract or otherwise direct what disposal shall be made of what will later be his dead body, because a dead body is not property. This principle has been upheld by many court decisions both in Great Britain and in the United States, some of the decisions being in state supreme courts in the twentieth century.

The two major deficiencies already mentioned regarding the Massachusetts law of 1831 were continued in these laws of the third phase, namely, that the laws were only permissive and not mandatory regarding the acts of civil officials, and that exceptions were made in regard to strangers, those who requested before death that they be buried and those for whom burial was requested by a kinsman or friend. This last exception was the most troublesome one. A friend might be an undertaker who wanted to be employed to conduct a funeral, or even a laborer who desired the job of digging a grave. These conditions were later remedied in some states by amendments that restricted kinsmen to spouses or those related by blood, and friends to those who had been legal representatives of persons before their death. The exception of strangers was also later limited by excluding from the group any person known as a vagabond or tramp.

The major weakness of the anatomical laws of the third phase was that they provided for discretionary action instead of mandatory action for civil officials in control of dead bodies that must be buried at public expense. Some of these officials had a personal aversion to dissection. Others disliked to make a decision that sent a body to the dissecting table because it brought criticism from those who had known the dead person in life. In some cases a decision to order a body for anatomical study was used to oppose the official rendering the decision for further elective office.

Opinions were expressed, and sometimes definite charges were made, that officials accepted or even solicited bribes for surrendering a dead body. This is the source of the provision in many modern anatomical laws for penalties against any official who accepts recompense for delivering a dead body for dissection, heavy fines, or in a few laws imprisonment, being prescribed.

A further difficulty was that from time to time an official favorable to surrendering bodies for dissection was replaced by one who was unfavorable, so that any institution might suddenly find a main source of supply of cadavers eliminated. All these conditions, and some others, made the supply of

cadavers from legal sources uncertain, sometimes extremely so, and at others more expensive than grave robbing, which therefore continued to supplement the uncertain and inadequate supply from legal sources.

The enactment of new anatomical laws in the sixties and seventies was not confined to New England. Similar laws appeared in states farther west, most of them a little later. Fourteen states had such laws before 1880. In all these the permissive feature predominated, although some of them had mandatory provisions regarding small categories, mainly the unclaimed bodies of those who died in state prisons.

*The Fourth Phase*

The fourth phase of the anatomical laws of New England began in the eighties. The conditions under the permissive type of law convinced all those engaged in the teaching of anatomy that the laws should be amended to substitute for the discretionary action mandatory directions to govern the actions of civil officials and employees in the surrender of dead bodies under their control.

Many factors entered the problem; a few will be mentioned. The relative influence of each of these varied in different states. Nearly all medical practitioners, remembering their own difficulties as students, were favorable to the change. Some of them were personal physicians of legislators and were able to influence them. A state medical society by resolutions and through a committee could influence the legislature of its own state as a body.

Between 1875 and 1890 the medical-education program changed greatly. The teaching session was lengthened. The requirement of attendance to be eligible for the degree of Doctor of Medicine was increased from two to three sessions. The graded curriculum was introduced, and along with it laboratory teaching in several subjects. This brought a different type of teaching in practical anatomy. The old method of hasty dissection of unembalmed bodies disappeared. Embalming was introduced and methods of storage of cadavers were much improved. This permitted acceptance of bodies from legal sources and their accumulation during the months that a medical college was not in session, and the former practice of completing a dissection in a week gave way to the spending of several weeks in the procedure, with much more attention to anatomic details. All medical colleges and their teachers favored a method that would give a more certain supply of cadavers and permit abandonment of the distasteful, illegal and uncertain reliance on grave robbing. Medical colleges as institutions and universities engaged in medical education were influential in inducing legislatures to institute reform.

Aside from the medical profession, medical teachers and medical teaching institutions, two other influences deserve mention. The majority of civil officials and employees disliked the duty under the permissive laws of deciding in each case whether to send a dead body to the dissecting table or to bury it at public expense. Whatever they did brought criticism, although from different sources. Mandatory laws would relieve them both of decisions and criticisms.

The aversion of the public to dissection had abated since earlier in the nineteenth century, when each detected case of grave robbing received publicity in the newspapers, which aroused public sentiment. The newspapers said little or nothing about the few detected episodes, and the great majority of grave robbings were not discovered because the technic of the procedure had become a fine art and the emptying of a grave seldom became known to the general public. Also, because the permissive laws made the surrender of dead bodies for dissection legal, the public came to believe that all bodies used for dissection were secured legally, a misconception nurtured by the medical profession.

These and other factors brought the support of legislators to amendments to the anatomical laws. After such amendment had been accomplished in one state it became easier to secure a mandatory law in a neighboring state.

Although Vermont did not join the four other states in New England that had medical colleges in enacting a permissive law about 1870, she led in passing an entirely mandatory law in November, 1884.[56] It directed that overseers of the poor in any town and superintendents of state institutions must, on written request of any licensed physician or surgeon resident in the state, surrender for the advancement of anatomical science any body that must otherwise be buried at public expense. The bodies of certain persons were excepted.[55] It was nine years before the next state in New England followed. Connecticut in 1893 enacted a similar mandatory law.[56] Then came corresponding laws in New Hampshire in February, 1897[57]; in Maine in March, 1897[58]; and in Massachusetts in 1898.[59] Rhode Island, never having had an anatomical law, did not enter into the fourth phase, just as she had not participated in the second and third phases. The enactment of these mandatory laws terminated the procedure of local grave robbing and the traffic in dead human bodies by importation from more distant states. These would have ceased long before if legislators had sooner followed the desires and advice of the medical profession.

## Provisions of Current Anatomical Laws in New England

Few amendments have been made to the anatomical laws of New England in the twentieth century. The discussion that follows is based on examination of the latest editions of the revised

statutes of Connecticut,[60] Maine,[61] Massachusetts,[62] New Hampshire[63] and Vermont.[64] The laws of each of these states for each year since the issue of the latest edition of its revised statutes have been examined for possible amendments. The use of the word "all" in what follows pertains to these five states only. Rhode Island, having no anatomical law, does not enter the discussion until the last paragraph of the section is reached. The keystone of all current anatomical laws is that the dead body concerned must be buried at public expense if it is not used for anatomic study. Certain exceptions will be mentioned later.

Members of a group of named civil officials and employees of political divisions have the duty to notify certain specified legal recipients when a body that must be buried at public expense comes under their control, and when the recipient has made a written request for the body they have the further duty of delivering it to him. Penalties are provided for failure in these duties. If no request is received, the body must be buried at public expense.

Kindred by blood or marriage, and in some states others, such as officers of fraternal organizations, may request that such bodies be buried at the expense of the claimant, and if such petitions are made within a specified time, varying in different states, they have priority over requests for the body to be used for dissection.

Three requirements are imposed on the recipient in all states. The body must be used only for advancement of anatomical, medical or surgical science or for medical education; its use must be confined to the state in which it is surrendered to the recipient; and after such use the remains must be decently buried. The recipient must give a bond that these requirements will be observed, in all states except Vermont, where a penalty is provided for nonobservance.

The phrase "decently buried" as applied to the remains of a dissected cadaver goes back at least to the charter granted to the College of Physicians of London by Charles II in 1663. None of the anatomical laws in New England define decent burial, as regards what is to be buried and where and when. No definition is given regarding what constitutes "remains," and whether these include only the skeleton or parts of it or the detached soft parts. No time of burial is specified. Some hold that this omission makes it legal to keep the remains or parts of them for an indefinite period of time before burial. An early law in one state specified that the burial must be made in a public cemetery, but this is omitted in later revisions. There is no provision that the remains of each cadaver shall be buried in a single excavation.

The law of Connecticut says that burial must accord with the rules of the State Board of Health. A specification in another relation in this statute uses the words "buried or cremated," which suggests that cremation is considered equivalent to burial, but there is no suggestion concerning whether the cremation must be in a licensed public crematory. Possibly court decisions or rulings of boards of health have defined decent burial, but so far as the anatomical laws go, the requirements are hazy.

The legal recipient of dead bodies destined for dissection varies in the different states. In Vermont, the recipient is any licensed physician resident in the state. In New Hampshire, the recipient is any licensed physician in the state, with preference given to such as are instructors in a medical school legally established within its borders. In Massachusetts, the recipient is the dean or other officer of any medical school legally established in the state. Connecticut is the only state in which the recipient is not a person or persons; there it is the Medical Department of Yale University. In Maine, the original recipient is any member of an anatomical board or the board as a whole. This board has the duty of distributing the bodies received to medical colleges or to persons who become the final recipients and use the body. This board was created by the law of 1897, and is the only one of its kind in New England. Originally the members were specified as those holding certain teaching positions in medical colleges in the state. The only medical college in the state was suspended in 1921. Ten years later the law was amended so that the superintendents of four named hospitals should constitute the membership of the board. The law in Maine is entitled "to provide for medical education"; in the amendment of 1931 premedical education and nursing education were specifically construed to be a part of medical education, and four colleges of arts in the state were named as legal recipients of dead bodies to conduct medical education under this construction.[65]

In only Maine and Connecticut does the anatomical law require any record of bodies received for dissection. In Maine, the record is made by the Anatomical Board, without specification as to what shall compose the record. In Connecticut, the recipient must make a record of the sex and, if known, the name and last place of residence. No descriptive record of stature, marks of identification or other details is suggested.

The exceptions to requirements of delivery for dissection of bodies that must otherwise be buried at public expense go back to early laws. In all states the body of a traveler or stranger who dies suddenly must be buried rather than be surrendered for dissection. Tramps, vagabonds and vagrants are excluded from this rule in Connecticut, Maine and New Hampshire. The anatomical laws of Massachusetts, New Hampshire and Vermont provide that the bodies of soldiers and sailors and of members of the Marine Corps who have served in any war of the United States and have been honorably discharged must be buried rather than delivered for dissection. In one state the widows

of these men are included. No mention of these restrictions is made in the law of either Connecticut or Maine, but in the former a law in another connection provides that men of these groups shall be buried at public expense.

The ancient provision still prevails in the anatomical laws of Connecticut, Massachusetts and Vermont that if a person requests in his last sickness that his body be buried, this must be done at public expense rather than have the body delivered for dissection. It has already been stated that the provision that a body must be buried at public expense if a kinsman so requested was a serious deterrent to the teaching of anatomy in the nineteenth century. This provision is still found in the law in Vermont.

Provision for the payment by the recipient of expenses connected with the transportation and burial of a cadaver is found in the anatomical laws of Vermont and Maine, but is not mentioned in those of other New England states.

Peculiar provisions are found in the laws of some of the states. The anatomical law of Connecticut states that the bodies of those dying from certain named contagious diseases shall not be available for dissection. The first disease named is Asiatic cholera, indicating the ancient origin of this section. A former law included those dying from unknown diseases because they might be contagious. It seems that such provisions belong in laws relating to the boards of health rather than in anatomical laws. The law also provides that the head of every educational institution that teaches anatomy must give a bond that no bodies illegally secured will be allowed to come within the premises under his direction. This law goes back more than a hundred years to the time when grave robbing was prevalent.

The provision regarding receiving the body of one who before death requested or consented to dissection of his body, which has already been mentioned as the first section of the law of 1869 in Maine, is still a part of the statute in that state. A physician who taught anatomy many years in Maine informs me that he knows of only one body that was received under this provision over a long period.

The most ancient relic in current anatomical laws in New England is in Massachusetts, where a judge may still order the body of an executed murderer to be dissected. This goes back to English common law before 1540.

All six of the states of New England still have laws against illegal disinterment of the dead, commonly called grave robbing, and impose penalties of fines or imprisonment or both. The fines extend to a maximum of $3000 in one state, and the maximum period of imprisonment to fifteen years in one state.

* * *

The history of the development of laws for the legal acquisition of dead human bodies for dissection in New England covers the more than three hundred years since its first settlement in 1620 and goes back at least another hundred years in the English common law. This history in the last one hundred and fifty years has included many endeavors and many disappointments on the part of the medical profession of New England.

The changes in this area were few until 1830. Four periods are found in the extension of legal provisions in New England: the first from 1641 to 1824, the second from 1831 to 1842, the third from 1869 to 1871 and the fourth from 1884 to 1898.

The few amendments made to the anatomical laws since 1898 indicate that teachers of anatomy in New England have felt reasonably secure in the legal acquisition of enough cadavers to meet their needs. The supply has varied with public economic conditions. One cannot predict future conditions, but recent diminution in the supply of cadavers is already causing anatomists to ponder on what the future will bring with increasing public provision for all classes of people, especially that class from which cadavers have usually been drawn.

The variations noted in the anatomical laws of the closely associated states of New England suggest that the anatomists should endeavor to formulate an anatomical law that includes the favorable parts of current laws and removes the unfavorable and redundant provisions. Such a formulation may become useful when opportunity is presented to amend and revise these laws, not only in New England but in every state of the Union.

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# INFECTIOUS MONONUCLEOSIS*

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INFECTIOUS mononucleosis, or glandular fever, was first described in 1889 by Pfeiffer.[1] The earliest report on the subject in this country was that by West[2] in 1896. The hematologic aspects of the process were extensively studied during the first three decades of this century and were crystallized by the work of Downey and McKinlay[3] in 1923. In 1932, Paul and Bunnell[4] observed that the serum of patients with infectious mononucleosis agglutinated the erythrocytes of sheep in concentrations above a normal titer. Since then there have been numerous reports on the disease, outstanding of which is the classic review by Bernstein.[5] In that monograph, the literature is reviewed and the vagaries of the disease are discussed in some detail. Numerous investigations have been made regarding the etiology of infectious mononucleosis, but no definite causal factor has yet been found.

Contratto,[6] of the Stillman Infirmary, Harvard University, has reported the largest single series to date, dealing with subject matter similar to that discussed in this paper.

The material for this report came from two sources: Dick Hall's House, the infirmary of Dartmouth College, and the Mary Hitchcock Memorial Hospital. The cases are those seen from January, 1935, to January, 1945. Both institutions are staffed by the same men, and all studies are done by the same laboratory. It was believed that a comparison of the two groups of cases, seen by the same physicians, would be of interest.

## INCIDENCE

Of the 141 cases in both institutions with a final diagnosis of infectious mononucleosis, 111 (82 in Dick's House and 29 in the hospital) were acceptable for study. The remaining 30 were discarded for various reasons, including inadequate records, insufficient data for diagnosis and missing laboratory studies.

The 82 infirmary cases represent 0.74 per cent of the total admissions for the period covered, and the 29 hospital cases represent 0.07 per cent. This difference is not surprising if one recognizes the specific age group that Dick's House serves. All the patients there were college students, save one who was a house officer of the hospital. In the hospital cases, it was interesting to note that 8 patients were from its nursing staff.

The yearly incidence shown in Table 1 roughly follows the number of general admissions for the year. The disease never occurred in epidemic proportions, although several such instances have been reported. Nor has the disease been seen in roommates, in spite of the fact that many of the patients were sick for several days before admission.

TABLE 1. *Number of Cases according to Years.*

| YEAR | DICK'S HOUSE | HOSPITAL | TOTALS |
|---|---|---|---|
| 1935 | 3 | 0 | 3 |
| 1936 | 5 | 2 | 7 |
| 1937 | 2 | 3 | 5 |
| 1938 | 8 | 1 | 9 |
| 1939 | 15 | 3 | 18 |
| 1940 | 14 | 6 | 20 |
| 1941 | 9 | 5 | 14 |
| 1942 | 11 | 3 | 14 |
| 1943 | 9 | 3 | 12 |
| 1944 | 6 | 3 | 9 |
| Totals | 82 | 29 | 111 |

Some observers believe that there is a seasonal incidence of this disease. This has not been clearly borne out in the present series, as evidenced by Table 2, which shows the incidence by months. The reason for the relative paucity of cases during the summer months is that until 1942 the college infirmary was closed from the latter part of June until the first part of September. The relatively high summer incidence in the hospital, on the other hand, is probably due to the influx of campers into the surrounding territory. Four of the hospital cases were in this category.

TABLE 2. *Number of Cases according to Months (1935–1944).*

| | DICK'S HOUSE | HOSPITAL | TOTALS |
|---|---|---|---|
| January | 12 | 2 | 14 |
| February | 8 | 2 | 10 |
| March | 8 | 0 | 8 |
| April | 8 | 1 | 9 |
| May | 10 | 2 | 12 |
| June | 7 | 6 | 13 |
| July | 1 | 6 | 7 |
| August | 3 | 3 | 6 |
| September | 6 | 2 | 8 |
| October | 5 | 2 | 7 |
| November | 5 | 2 | 7 |
| December | 9 | 1 | 10 |
| Totals | 82 | 29 | 111 |

Infectious mononucleosis is a disease of the younger age groups. Cases occurring in the sixties and even one at the age of seventy have been reported. In the Dick's House series, the average age was 20.2 years, with the youngest patient seventeen years old and the oldest thirty-one. In the hospital there was a greater spread in the age incidence, the youngest patient being ten years old and

*Presented at the annual meeting of the New Hampshire Medical Society, Manchester, May 15, 1945.

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the oldest forty-six, with 5 patients over thirty. The average age, 22.6 years, was slightly higher than that in the infirmary group.

The sex distribution was naturally irregular. All the Dick's House patients were men, whereas in the hospital group there were 18 females and 11 males. Previous general studies have shown a slight preponderance of males.

## Clinical Manifestations

The protean nature of infectious mononucleosis has already been mentioned, and a review of the symptomatology confirms this. In Table 3 the complaints, both major and secondary, noted on admission in the combined series are listed. This table shows the most frequent symptoms, which are nonspecific. Sore throat was by far the outstanding single complaint and was evident in 65 cases on admission. During the course of their illness several more patients developed this symptom, so that by discharge 83 patients had complained of it at some time. The degree of soreness

Table 3. *Major Symptoms in Both Series of Cases.*

| Symptom | No. of Cases |
|---|---|
| Sore throat | 65 |
| Grippal symptoms | 52 |
| Headache | 35 |
| Fatigue | 16 |
| Lymphadenopathy | 15 |
| Fever | 13 |
| Chills | 11 |

varied from mild dryness or scratchiness to severe pain with inability to swallow. It is worthy of note that in a number of cases the sore throat at the onset was unilateral. Many of these patients developed a typical Vincent's type of ulcer.

The grippal symptoms included malaise, aches in the arms and legs, backache and neck ache.

Headache was the next most striking single symptom; it was frequently the only complaint. It varied from severe to quite mild, and was usually generalized or frontal. In no case was meningitis suspected, and no lumbar punctures were done.

In most cases the presenting symptom of fatigue had been present for some time. One student entered with this as his only complaint. He had become aware of it by observing his progressively decreasing ability to keep up with his mates on the swimming team.

Feverishness and chills occurred and the chills were often true rigors, especially in cases with marked throat involvement.

The rest of the complaints have been grouped by systems (Table 4). Under gastrointestinal symptoms are included nausea, vomiting, diarrhea and abdominal pain. The last-named was usually associated with diarrhea or vomiting, but in 4 cases it was in the left upper quadrant and was undoubtedly related to splenic change. In 1 case the spleen had ruptured.

The respiratory symptoms ranged from a mild cold to a severe cough with manifestations suggestive of pneumonia.

The neuromuscular symptoms were comparatively unrelated, consisting of dizziness, nervousness, drowsiness, mental confusion — in a patient who became frankly delirious — and pain in the arm.

Table 4. *Symptoms by Systems in Both Series of Cases.*

| System | No. of Cases |
|---|---|
| Gastrointestinal | 28 |
| Respiratory | 19 |
| Neuromuscular | 8 |
| Cutaneous | 4 |
| Ocular | 5 |
| Cardiovascular | 2 |
| General | 6 |

The skin changes consisted of boils or a rash.

The eye symptoms were unusual. Three patients presented watery eyes as the only early complaint, and 2 others noted marked puffiness of the lids.

Two patients entered with tachycardia as the only symptom.

The general symptoms were confined to 4 patients who complained of weight loss and 2 who on admission had no complaints referable to the disease and were hospitalized because of skiing injuries. In the latter cases, lymphadenopathy was discovered on the admission physical examination and the disease was diagnosed after blood-cell counts and agglutination tests had been reported.

## Physical Signs

The throats of only 25 patients in both groups were considered normal on admission. The remainder showed varying degrees of inflammation. Thirty-two exhibited exudate on the lymphatic tissue of the tonsils or pharynx. Six showed frank ulceration of the fauces or tongue. One patient had a full-blown peritonsillar abscess, and another developed a lingual abscess later in the disease. Petechial areas on the soft palate, quoted by several authors as a fairly frequent finding, were present in 3 cases.

Smears and cultures were done in 49 cases. A beta-hemolytic streptococcus was isolated in 7 of these; Vincent's organisms were observed in 22; and a mixed infection was present in 20.

Marked gingival involvement was seen in only 2 cases, being accompanied in both by a pharyngeal infection of the Vincent's variety. Because of the frequent association of Vincent's infections and infectious mononucleosis, any case in which the diagnosis of Vincent's tonsillitis or gingivitis is made should be watched closely, with repeated studies, to exclude a possible underlying infectious mononucleosis.

Fever was absent in only 3 cases in both groups. The average temperature on admission was 100.6°F. in the hospital group and 100.4°F. in the infirmary group. The highest temperature recorded was 105°F. in Dick's House (2 cases) and 104°F. in the hospital.

The mean duration of fever was 5.9 days in the hospital group and 7.2 days in the infirmary group. In the latter, 2 patients were febrile for twenty days and 1 for nineteen days, whereas the longest duration of fever in the hospital was fifteen days. The fever follows no orthodox pattern and may return after an early subsidence usually with the onset of some complication.

The lymph-node enlargement that is characteristic of this disease and gives it the name "glandular fever" was pronounced in some cases and minimal or absent in others. Table 5 shows the extent and degree of involvement in the two groups. As a rule, the nodes are firm, discrete and either nontender or very slightly tender. Occasionally with severe throat infections the tonsillar nodes are exquisitely tender.

TABLE 5. *Lymphadenopathy.*

| LOCATION | DEGREE OF ENLARGEMENT* SLIGHT | MODERATE | MARKED | TOTAL NUMBER OF CASES |
|---|---|---|---|---|
| Anterior cervical | 5 | 72 | 13 | 90 |
| Posterior cervical | 2 | 42 | 6 | 50 |
| Axillary | 0 | 32 | 3 | 35 |
| Inguinal | 0 | 28 | 2 | 30 |
| Epitrochlear | 0 | 11 | 1 | 12 |
| Preauricular | 0 | 1 | 0 | 1 |

*"Slight enlargement" signifies nodes slightly under 1.0 cm. in diameter; "moderate," up to 1.5 cm.; and "marked," 2.0 cm. or greater.

In 9 cases, — 3 in the hospital and 6 in the infirmary, — there was no demonstrable lymphadenopathy, but in 3 of these there was a definitely palpable spleen. Ten cases showed lymphadenopathy elsewhere than in the anterior cervical chain, which was involved alone in 35 cases. In 2 cases in each group, there was isolated enlargement of the posterior cervical chain. In no case was isolated axillary, inguinal or epitrochlear enlargement found, nor was there any positive evidence of mediastinal or mesenteric lymphadenopathy.

The incidence of splenomegaly in infectious mononucleosis varies greatly in the published reports, with an average of 50 per cent of all cases showing this important sign. Press, Shlevin and Rosen[7] report the incidence in a recent series as 72 per cent. There was a palpable spleen in 15 cases (53 per cent) in the hospital group and in 33 cases (40 per cent) in the infirmary group. It is of interest that the percentage was considerably higher in the last half of the study, from 1940 through 1944 — 58 per cent and 59 per cent, respectively. This probably represents no change in the disease but rather gives evidence of a more careful search on the part of the staff and the house officers.

The spleen in most cases was firm and nontender and was felt with difficulty, although several cases demonstrated enlargement 4.0 cm. below the left costal margin. In 4 cases, there was distinct tenderness in the splenic area.

Jaundice occurred in 3 cases, — 2 in the infirmary and 1 in the hospital, — but in 1 of the former there had been pre-existing chronic biliary infection. In the other cases the jaundice was moderate, with icteric indices of 24 and 29, respectively. Six other cases were reported as appearing jaundiced, but the icteric index was normal.

Associated with the sallow, waxen color that was mistaken for jaundice there was a mild facial edema, occurring for the most part about the eyes. This was present in 5 of the students and in 1 of the hospital cases. In 2 cases the appearance was striking enough to suggest the diagnosis of acute nephritis or trichinosis.

Cutaneous changes were evident in 9 cases. In the hospital group (2 cases), an extremely ill patient developed a generalized maculopapular eruption that rapidly became hemorrhagic. The other patient showed multiple ecchymoses, unassociated with thrombocytopenia or some other blood anomaly. These cleared spontaneously. Five of the infirmary patients showed a faint erythematous rash on the trunk and arms, which lasted for only a day. The others had a generalized maculopapular eruption for two days and were at first thought to have rubella.

Neurologic manifestations other than headache were infrequent. None were present in the hospital group, and in the infirmary they were seen in only 2 patients. One of them was the patient previously mentioned who became delirious at the height of the fever. The other entered with the complaint of "neuritis" in the left arm, which was actually a pain in the ulnar aspect of the forearm. The only finding was that of several large lymph nodes in the left axilla. These promptly disappeared, and with them the discomfort.

### BLOOD FINDINGS

Examination of a stained blood smear is the simplest method of establishing the diagnosis in a suspected case of infectious mononucleosis. The characteristic picture shows a moderate leukocytosis, with a mononuclear percentage above 60. The mononuclear cells consist of normal monocytes, normal lymphocytes and the so-called "toxic lymphocyte," which is typical of the disease. The last-named cell makes up the majority of the mononuclear elements. It has certain distinct characteristics. It is variable in size, shape and staining properties. The nucleus, whatever its shape, is usually stained darkly and coarsely. The cytoplasm may be pale blue but is oftener deeply basophilic and vacuolated, presenting a foamy appearance. Since there was no significant variation in the blood picture in the two groups, they will be discussed together.

The average white-cell count on admission was 10,600, only a slight increase over normal. The highest admission count and also the highest count throughout the illness was 33,600. Not infrequently in the early stages there is a pronounced leukopenia. Eleven counts were below 4000 and two were below 2000 (1700 and 1400, respectively). As a rule, th patients who exhibited leukopenia on admission were sicker than those with a higher white-cell count. They showed a gradual increase in the white-cell count as the disease progressed. There were 9 cases with counts of over 25,000.

On admission the average percentage of polymorphonuclear leukocytes was 39. Later this fell to between 20 and 25 per cent. The disappearance of these cells may be alarming. Nine cases showed 15 per cent or less, and 1 showed only 4 per cent. The increase in mononuclear cells, which was both relative and absolute, roughly paralleled the clinical course.

Other abnormalities noted in the smears in 9 per cent of the cases included basophilic stippling of the erythrocytes and young forms of the polymorphonuclear series, which in 1 case had led to an earlier diagnosis of leukemia.

The necessity for taking frequent blood smears should be mentioned. It is not unusual to find the white-cell count relatively normal early in the disease and to note a change after the patient has apparently recovered.

The development in 1932 of the diagnostic test by Paul and Bunnell[4] — variously known as the sheep-cell agglutination test, the heterophil-antibody test and the Paul–Bunnell test — has provided a valuable diagnostic aid. This relatively simple procedure is a measure of the ability of the serum to agglutinate washed sheep erythrocytes in increasing dilutions of serum. The titer thought by most investigators to be diagnostic is 1 : 64.

One or more agglutinations were performed in 95 cases of the combined series. A positive reaction was present in 86 (90 per cent). In the 9 cases in which the reaction was negative and in the remaining 16 cases, blood smears and clinical findings were so typical that repeated tests would no doubt have given positive reactions. In many of the cases that finally showed a positive reaction, negative or low titers were encountered early in the course of the disease. On the other hand, one patient on the second day after admission had a positive agglutination reaction in a dilution of 1:114,000, and on the twelfth day this was still positive in a dilution of 1:7000. This patient was a moderately allergic person who had had no recent injections that could have accounted for such a titer of antibody.

No case had a history of recent serum disease, the only other common process that produces a positive heterophil reaction. One case had been diagnosed elsewhere as brucellosis and had been treated with injections of brucellin. The brucellar agglutination that was positive in a dilution of 1:160, whereas the heterophil test was positive in a dilution of 1:896. The blood smears were typical of infectious mononucleosis.

Positive agglutination reactions for typhoid and paratyphoid B bacilli in a dilution of 1:160 were found in still another patient, in whom the diagnosis was for some time obscure. This peculiarity of the serum of patients with infectious mononucleosis has been noted by other investigators and may, unless recognized, make matters confusing for the physician.

### Complications

Besides the previously mentioned cases of peritonsillar and lingual abscess, there were various other complications in this series.

One patient developed acute purulent otitis media on the fourth hospital day, which responded to paracentesis and chemotherapy. Although cough was a frequent symptom, only 1 case showed bronchopneumonia. Another patient, however, had been in Dick's House for sixteen days because of infectious mononucleosis and had made an apparently normal recovery. He was readmitted eight days later because of findings suggestive of atypical or virus pneumonia and died on the eleventh day. It was in this case that the polymorphonuclear count dropped to 4 per cent, although by the time of the second admission it had risen to 52 per cent.

None of the patients who complained of abdominal pain presented the clinical picture of acute appendicitis. Except for 1 case, there was no major gastrointestinal complication. This patient, who was moderately sick, had several episodes of hematemesis over a twenty-four-hour period. Tarry, guaiac-positive stools were found for several days thereafter. He improved promptly on general supportive measures, and no specific cause for this striking manifestation was ever discovered.

Perhaps the most unusual complication in the series was that of a nineteen-year-old student who in February, 1936, was admitted to Dick's House shortly after falling and injuring his left side in a skiing accident. Prior to this, he had been in apparently good health. On admission, he complained of pain in the left upper quadrant of the abdomen, radiating to the left shoulder. Examination showed the typical findings of a ruptured spleen. The white-cell count was 4250, with 89 per cent mononuclear cells. After transfusion, an exploratory celiotomy was done. A large spleen weighing 970 gm. and with a jagged laceration was removed. The postoperative course was stormy, being complicated by atelectasis and pneumonia, but the patient eventually made a normal recovery. The preliminary diagnosis from the microscopic sections was lymphoblastoma of the Hodgkin's type, but the blood smears, re-examination of the spleen by several pathologists and the subsequent course

have borne out the diagnosis of infectious mononucleosis. At present, the patient is in excellent health. Rupture of the spleen in infectious mononucleosis has been reported previously.

Lymph-node biopsy is rarely either necessary or helpful in infectious mononucleosis. In a patient who entered with complaints of pain in the left upper quadrant of the abdomen and low-grade fever and in whom axillary and inguinal lymph adenopathy and splenomegaly were found, an inguinal node was excised for diagnosis. Microscopic examination showed a normal node. The patient subsequently developed jaundice and was seen in consultation by Dr. William P. Murphy. The diagnosis of familial hemolytic jaundice was entertained until the late appearance of several strongly positive heterophil reactions. The patient had a protracted convalescence and after eight months still had a palpable spleen.

Tachycardia, an admission complaint in 2 cases, disappeared on bed rest. No other cardiac complications were discovered, and further study of these cases showed no abnormality. Urinary complications, except for transient albuminuria, were not seen.

Although not a complication in the usual sense of the word, persistent fatigue or weakness and inability to regain former endurance was a feature in many cases. Three patients were readmitted because of a relapse or persistence of symptoms. One, a nurse, was hospitalized three times.

### Length of Hospitalization

The number of days spent as inpatients was similar in both groups, averaging 13.1 days in the infirmary and 11.4 days in the hospital. The shortest time for either was two days, and the longest was thirty-six days in the hospital and forty days (2 cases) in the infirmary. Eighteen patients were hospitalized for over twenty days. The nurse mentioned above aggregated forty-two days for her three admissions.

The duration of hospitalization is not a fair estimate of the duration of illness, for many patients were dismissed to their homes for convalescent care, and the first hospital day usually came some time after the first day of the illness.

### Treatment

With the etiology of infectious mononucleosis unknown, there can be no specific therapy. Intelligent handling and the use of general measures is adequate for the average case. Sore throat was treated by saline-solution irrigations, and pain was controlled by codeine and aspirin. Mouthwashes or gargles with sodium perborate were useful in cases with Vincent's infection. Their routine use seems worth while in preventing gum involvement. Neoarsphenamine was rarely used and appeared to produce no dramatic results. Sulfadiazine had no appreciable effect on the disease.

Cases in which vomiting, diarrhea, sweating or dysphagia seriously upset the fluid and electrolyte balance profited by parenteral fluids.

### Summary

The history and clinical findings in two groups of cases of infectious mononucleosis, occurring at the same time and treated by the same physicians, are discussed, and significant features are described.

Infectious mononucleosis is a fairly frequent, usually benign disease and is most frequently seen in young adults.

The diagnosis of infectious mononucleosis may be simple or obscure. A high degree of suspicion, not only in cases with symptoms suggestive of the usual upper respiratory infections but those resembling typhoid fever, leukemia, brucellosis, Hodgkin's disease, nephritis and infectious jaundice, will lead to the hematologic and serologic studies necessary to establish such a diagnosis.

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**Correction.** In the paper "The Rehabilitation of Patients Totally Paralyzed Below the Waist: With Special Reference to Making Them Ambulatory and Capable of Earning Their Living. I. Anterior Rhizotomy for Spastic Paraplegia" by Dr. Donald Munro, which appeared in the October 18 issue of the *Journal*, the word "SPIRAL" in the heading in the first column on page 454 should be changed to "SACRAL."

# MEDICAL PROGRESS

## HEMOGLOBINEMIA AND THE HEMOGLOBINURIAS (Continued)*

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### PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

Paroxysmal nocturnal hemoglobinuria, the strangest of this entire group of bizarre diseases, is also the most infrequent. Only 50 cases have been reported since Marchiafava and Nazari[60] first recognized the condition as a separate disease entity in 1911. The disease usually appears during the third or fourth decade of life, although it has been described in a child of five and a half years[61] and a woman aged fifty-two years.[62] Of the reported cases, 25 have been males and 25 females. There is no demonstrable hereditary or familial tendency, and careful studies have failed to reveal predisposing diseases or etiologic factors of any type. The victims of the disease usually have been perfectly well prior to its onset, which may be insidious, with gradual progression of symptoms for several years, or abrupt, with the sudden onset of massive hemoglobinuria. The hemolytic process at first may be so mild that the only symptoms are those of hemolytic anemia and hemoglobinuria may not develop until years later.[63] Once established, the disease is remarkable for its chronicity, its spontaneous remissions and exacerbations and its refractoriness to all therapeutic measures. Although cases have been observed for as long as thirty-three years,[64] no case of complete recovery has been recorded. The clinical picture is one of persistent and marked chronic hemolytic anemia with superimposed exacerbations, during which the hemolytic process is accelerated and hemoglobinuria appears. In the intervals between exacerbations, anemia is always present, with its associated symptoms of weakness and easy fatigability. Hemoglobinemia can always be demonstrated, and mild jaundice is frequently noted. During the intervals between acute exacerbations patients usually are able to perform light work, and once they have become adjusted to their anemia they get on remarkably well.

Exacerbations occur at irregular intervals and usually without obvious cause, although the onset of any infection, no matter how mild, may precipitate an attack. Exertion and exposure to cold neither precipitate nor aggravate attacks. They may occur at frequent intervals, or rarely, many months or several years[64] may intervene between attacks. Each attack may persist for only a few days or may last for several weeks. With the onset of an exacerbation, the hemolytic process increases in severity, anemia becomes more marked, hemoglobinemia rises and hemoglobin appears in the urine. During these attacks the patient usually is markedly prostrated and completely incapacitated. Chills and fever may occur, vague aches and pains are noted, and severe cramping abdominal and lumbar pains are occasionally present. The spleen and liver are frequently moderately enlarged and may vary in size from time to time.

One of the most striking features of the disease is the nocturnal character of the hemoglobinuria. During the daytime waking hours the urine may be completely free of hemoglobin, but at night and shortly after waking in the morning the urine may contain so much hemoglobin that it is absolutely black in color. As subsequently noted, this apparent diurnal variation in hemoglobinuria is really a reflection of the sleeping and waking habits of the patient, the actual cause of the increase in hemoglobinuria at night being associated with increased hemolysis occurring during sleep.

Venous thromboses have occurred in approximately one quarter of the reported cases and constitute the most serious complication of the disease. They usually develop during the acute exacerbations but also have been observed during periods of remission when there was no hemoglobinuria.[65] They most frequently occur in the portal system, with involvement of the mesenteric veins and ensuing intestinal infarction.[62, 66] Intracranial vascular occlusions with hemiplegia are also of fairly common occurrence.[62, 65] Thromboses of the peripheral veins and pulmonary infarction also have been reported.[63, 66–68]

Why thromboses are prone to develop in this disease has not been satisfactorily explained. Scott, Robb-Smith and Scowen[62] have suggested that localized accumulations of erythrocyte stroma or agglutination of hemolyzing erythrocytes may produce vascular occlusion. Hjärre[69] demonstrated that injections of erythrocyte stroma into animals produced thromboses in the lungs and areas of focal necrosis in the liver — another lesion frequently encountered in paroxysmal nocturnal hemoglobinuria. It is possible that a similar mechanism is operative in paroxysmal nocturnal hemoglobinuria, but some other factor is probably present as well, since other types of hemolytic anemia are not characteristically associated with thrombotic manifestations.

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Free hemoglobin is constantly present in the blood plasma of patients with paroxysmal nocturnal hemoglobinemia, although its concentration may be below the renal threshold and none may be present in the urine. When the total concentration of heme pigments is in excess of 250 to 300 mg. per 100 cc., methemalbumin appears and may be detected spectroscopically. Plasma bilirubin always is increased and, following excessive hemolysis, may reach levels of 5 or 6 mg. per 100 cc., with associated jaundice.

In addition to these signs of increased blood destruction, there is also evidence of an increased rate of blood regeneration: reticulocytosis is constantly present during periods of exacerbation and may reach levels of 20 or 30 per cent. The bone marrow shows increased erythropoietic activity, with hyperplasia of erythroblastic elements.[62] In spite of the marked reticulocytosis and increased blood regeneration, the rate of blood destruction is always in excess of blood formation, and a marked anemia is constantly present. As a rule, the red-cell count varies between 2,000,000 and 3,000,000, but a value of 1,000,000 is frequently encountered and it rarely rises to 4,000,000. The erythrocytes are moderately macrocytic, and the mean corpuscular hemoglobin concentration is normal. They show a normal resistance to hemolysis by hypotonic solutions of sodium chloride, and the Donath–Landsteiner test for cold hemolysins is negative.

Diagnostic of this disease is the increased susceptibility of the red blood cells to hemolysis by acidified plasma. This abnormality was first noted in certain cases of hemolytic anemia by Van den Bergh,[70] but he did not succeed in distinguishing the disease entity of paroxysmal nocturnal hemoglobinuria from other forms of hemolytic anemia. Ham[71, 72] and Dacie, Israëls and Wilkinson[73] recently reinvestigated the sensitiveness of these erythrocytes to increased acidity. Ham's studies have placed the so-called "acid hemolysis test" on a sound basis and have done much to clarify the pathologic physiology of the disease. He found that marked hemolysis was produced when the patient's erythrocytes were mixed with blood serum obtained either from the patient or from a normal control subject and when the acidity of the mixture was increased to pH 6.8 or 7.0 by equilibration with carbon dioxide or by admixture with a small amount of mineral acid. Normal control erythrocytes showed no or minimal hemolysis when similarly treated. These studies indicated that the fundamental abnormality resided in the erythrocytes but did not exclude the possibility that the cells might have been sensitized by absorption of some hemolysin. Subsequent investigations revealed no plasma hemolysin or antibody of any type but did demonstrate that a constituent of normal plasma was essential for the production of hemolysis, even in acid mediums.[74] This plasma substance was extremely thermolabile, being destroyed by exposure to a temperature of 56° C. for five minutes, and it was concluded that it was closely associated with, if not indistinguishable from, complement or alexin. In contrast to the hemolytic system existing in cold hemoglobinuria, the hemolytic activity of the inactivated serum could not be restored by the addition of guinea-pig complement.

Although the increased susceptibility of erythrocytes to acid hemolysis is a characteristic feature of paroxysmal nocturnal hemoglobinuria that has been confirmed by numerous investigators, it is questionable whether a similar mechanism operates to produce intravascular hemolysis in patients with disease. Ham[72] clearly demonstrated that the nocturnal character of the hemoglobinuria was merely a reflection of the fact that hemolysis is accentuated and hemoglobinuria occurs during sleep. By causing patients to sleep during the daytime and stay awake at night, hemoglobinuria could be made to appear during the daytime, while the urine was free from hemoglobin during the night. By preventing sleep for many hours, hemoglobinuria was eliminated, only to recur as soon as the patient went to sleep. Ham attempted to correlate these observations with the slight decrease in the alkalinity of the blood that is stated to occur during sleep.[75] Pulmonary ventilation decreases during sleep, and there is a consequent slight increase in the carbon dioxide content of arterial blood, with a resultant decrease in the alkalinity of the plasma. These changes seldom, if ever, lower the reaction to pH 7.2, however, and in several carefully studied patients with paroxysmal nocturnal hemoglobinuria, it was impossible to demonstrate any change whatever in the reaction of the blood during sleep.[72, 76] Ham has suggested that the reaction of the blood in the viscera may fall to levels low enough to produce marked hemolysis, but there is no experimental support for this hypothesis. There is no doubt of the fact that, in vitro, a slightly acid blood plasma produces hemolysis of the erythrocytes in the presence of human complement. Whether this mechanism operates in vivo is still open to question. At present, no better hypothesis than Ham's has been advanced. A somewhat analogous situation exists in familial hemolytic jaundice: the erythrocytes in this disease show an increased susceptibility to hemolysis by hypotonic solutions of sodium chloride, and yet there is no evidence that any condition of hypotonicity exists in vivo to account for the increased hemolysis in patients with this disease.

Leukopenia and thrombocytopenia are characteristic features of paroxysmal nocturnal hemoglobinemia. The white-cell count is usually between 2000 and 4000, and the differential count is relatively normal or shows a relative lymphocytosis. The leukopenia may contribute to the susceptibility and poor resistance of these patients to infections, al-

though during acute infections the leukocyte count often rises to high levels.[65] The blood platelets are markedly reduced in number, usually ranging between 100,000 and 150,000 per cubic millimeter. Several cases have shown hemorrhagic tendencies, which are probably attributable to the thrombocytopenia.[61, 62, 77] Following splenectomy, the number of leukocytes and platelets may increase to normal, but occasionally severe leukopenia and thrombocytopenia persists indefinitely.[65]

Hemoglobinuria, perhaps the most prominent feature of the disease, has attracted much attention to the kidneys of patients with paroxysmal nocturnal hemoglobinuria. Before the recognition of the fact that hemoglobinuria was merely a reflection of an elevated concentration of hemoglobin in the blood plasma, unsuccessful operations were performed on the kidneys of several patients in an attempt to explain and remedy the hemoglobinuria.[78] Although it now is recognized that the role of the kidneys is probably secondary, it still is of considerable interest to determine what effect the excretion of large amounts of hemoglobin might have on renal function. Oddly enough, relatively few adequate studies of renal function have been made in patients with paroxysmal nocturnal hemoglobinuria. Ham has recorded a reduction in urea clearance in 3 cases, but in one of these patients there was coexisting pyelonephritis, which eventually proved fatal. Other investigators have limited their discussion of renal function to the comment that the urine occasionally contained albumin and that hemosiderin was constantly present.

Recent studies of renal blood flow, glomerular filtration and tubular function in 2 patients with severe paroxysmal nocturnal hemoglobinuria revealed that renal function was remarkably good in spite of the fact that hemoglobinuria was almost constantly present.[79] The abnormalities encountered were almost identical with those found in other patients with severe anemia and were believed to be a reflection of the anemia itself and not of the hemoglobinuria.

These observations are of considerable importance, since they support the theory that excretion of hemoglobin per se is relatively harmless and that the anuria and uremia resulting from mismatched transfusions and crushing injuries are due to some cause other than hemoglobinuria.

Although hemoglobinuria occurs irregularly, hemosiderin is excreted at all times in the urine of patients with paroxysmal nocturnal hemoglobinuria, and as Marchiafava[80, 81] first emphasized, this hemosiderinuria is even more typical of the disease than hemoglobinuria. With the exception of hemochromatosis, paroxysmal nocturnal hemoglobinuria appears to be the only disease in which excessive amounts of iron are continually excreted in the urine. Granules of iron-containing pigment can be demonstrated readily in the urinary sediment at all times simply by mixing the sediment with a small amount of dilute ammonium sulfide and observing the black granules of ferrous sulfide with the microscope. As a rule, these granules lie within renal epithelial cells or within cellular casts.

At autopsy, patients who have succumbed to paroxysmal nocturnal hemoglobinuria are found to have a characteristic distribution of iron-containing pigment in their tissues, a distribution that is found in no other disease entity. The kidneys are described as "snuff-brown"[62] or mahogany in color, and the Prussian blue reaction for iron-containing pigment is strongly positive in the cortices and interpyramidal substance but negative in the pyramids. The reaction is also negative when applied to the liver and to the spleen. Histologic examination reveals large amounts of iron-containing pigment, presumably hemosiderin, in the epithelial cells of the renal tubules but none elsewhere. In the proximal convoluted tubules the hemosiderin is small in amount and finely dispersed; in the ascending loops of Henle and in the distal convoluted tubules, large amounts of iron are present in coarse granular aggregates. Examination of the liver, spleen and bone marrow shows either no iron or a less than normal amount. This distribution of iron in the tissues is peculiar to paroxysmal nocturnal hemoglobinuria. In other forms of chronic hemolytic anemia, for example, familial hemolytic jaundice, the liver, spleen and bone marrow, as well as the kidneys, contain large amounts of iron. In hemochromatosis, iron is deposited in the adrenal cortices, the testes and the skin, in addition to the spleen, liver and kidney.

The unusual distribution of iron in these cases is readily explained by a consideration of the pathologic physiology of hemoglobinemia and of iron metabolism in anemia. The persistent hemoglobinemia results in an almost constant leakage of hemoglobin through the glomerular membrane into the glomerular filtrate. As a consequence, tubular epithelial cells must constantly be reabsorbing hemoglobin, breaking it down to hemosiderin and returning it to the hematopoietic system for reuse in the manufacture of new hemoglobin. This explains why deposits of iron are readily demonstrated in the tubular epithelial cells and why there is a persistent hemosiderinuria. When these cells become "loaded" with hemosiderin they may be dislodged and excreted in the urine. In contrast to the situation in the kidneys, the chronic anemia and the constant demand for iron for hemoglobin synthesis results in a steady drain on the usual sites of iron storage. These tissues consequently contain less than their usual complement of iron.

The treatment of paroxysmal nocturnal hemoglobinuria has been quite unsuccessful. Since the erythrocytes of afflicted subjects have been shown to be susceptible to hemolysis by acid mediums, attempts have been made to alkalinize the blood

and urine by the administration of sodium bicarbonate and other bases. Although transient benefit has been reported in some cases,[72, 76] hemoglobinuria soon reappeared and exacerbations were likely to be much severer than when alkalies had not been administered. Frequent feedings during waking and sleeping hours with the hope of producing an alkaline urinary tide have not been beneficial.[72, 77] Injections of sympathicomimetic and parasympathicomimetic drugs have been reported to be of value by Hoffman and Kracke,[76] but the effects obtained are far from clear cut.

Splenectomy has been performed in many cases of paroxysmal nocturnal hemoglobinuria, always without benefit, and numerous fatalities have resulted from the effects of the operation.

Blood transfusions used as a supportive measure when anemia is extreme have definite but transient therapeutic value. Patients with paroxysmal nocturnal hemoglobinuria are prone to develop severe pyrogenic reactions after transfusions with compatible blood, and hemolysis of the patient's own erythrocytes may be greatly accentuated after transfusion. Thromboses may also occasionally occur during or after transfusion. In spite of these hazards, however, it is necessary to transfuse some patients at fairly regular intervals to combat the anemia, which otherwise becomes so extreme that it completely incapacitates the patient.

Although transfusion of blood may temporarily aggravate the hemoglobinuria, a definite remission of the hemolytic process often occurs and the urine may remain free from hemoglobin for a period of weeks or months following the injection of the blood.[65, 82]

In summary, there is no specific therapy for paroxysmal nocturnal hemoglobinuria, but with careful medical supervision and the judicious use of blood transfusions, patients with this disease can lead useful lives for many years. Operative procedures of all types, especially splenectomy, are to be avoided, and any infection, no matter how slight, should be immediately controlled.

## Blackwater Fever

Certain cases of falciparum malaria are complicated by acute hemolytic anemia, hemoglobinemia and hemoglobinuria, a syndrome recognized for centuries and commonly known as "blackwater fever." The syndrome occurs most frequently in Europeans who have resided several years in highly endemic malarial regions and who have experienced several attacks of falciparum malaria. The disease is usually precipitated by medication with quinine,[63] atabrine[84] or plasmoquine,[85] and is characterized by rigor, fever, vomiting, jaundice and the passage of black urine containing large amounts of hemoglobin. Prostration and extreme weakness are outstanding features of the disease and are a reflection of the rapidly progressing anemia produced by the excessive hemolysis. The number of erythrocytes may decrease to 20 or 30 per cent of normal within twenty-four to forty-eight hours, and the deleterious effect of this sudden and extreme anemia on the heart and vascular system may be so severe that the slightest exertion may precipitate sudden death. Blood destruction is occasionally so excessive that shock-like states develop and death may occur within a few hours.

Hemoglobinuria is intense and may persist for a few hours or several days. Black urine lasting for longer than twenty-four hours is of grave prognostic significance.[83] Some patients develop oliguria, anuria, and uremia following the hemoglobinuria, a sequence of events almost always leading to death regardless of therapeutic measures.

Treatment of the syndrome is limited to supportive measures, which must be instituted as soon as evidence of increased hemolysis is apparent. Warmth, maintenance of adequate fluid and electrolyte intake, sedation and absolute bed rest must always be provided. Victims must not be moved until the acute episode is over, because of the danger of precipitating sudden death from fibrillation of the weakened cardiac ventricle. Blood transfusions should be administered if the anemia is rapidly progressive or of extreme severity. Transfusions are of considerable benefit in the majority of cases, although the transfused cells are apparently hemolyzed just as readily as the patient's own erythrocytes.[86]

Antimalarial drugs should be administered to patients with blackwater fever only if there is unmistakable evidence of heavy parasitization of erythrocytes, an occurrence which is very infrequent.[87] Some authorities warn against giving antimalarials at any time during the acute course of the disease since it has been suggested that these drugs aggravate the hemolytic process.[88, 89] Foy and Kondi,[90] from experience gained from 450 cases of blackwater fever in Macedonia, advise cautious administration of quinine or atabrine if parasites are particularly numerous and if malarial paroxysms are superimposed on the symptoms of blackwater fever.

Since the investigations of Baker and Dodds[21] indicated that an alkaline urine might prevent the formation of acid hematin casts in the renal tubules, it has been standard practice to attempt to alkalinize the urine of blackwater-fever patients. Although recent authoritative publications[24, 89] still recommend the administration of alkalies until the urine becomes alkaline, careful consideration of experimental and clinical evidence gives little support to the belief that alkalinization changes the course of the disease or decreases the number of fatalities.[34] Large quantities of alkali may actually be harmful, since they tend to upset further an already disturbed acid-base balance and produce alkalosis of severe degree. In the administration of fluid

and electrolytes the wisest course to follow appears to be the maintenance of as normal a sodium, chloride and carbon dioxide concentration in the plasma as possible. The urine probably should be alkalinized if this can be achieved without unduly disturbing the plasma-electrolyte equilibrium, but alkalies should never be forced to the point of producing alkalosis.

The cause of blackwater fever has been the subject of much speculation since the first clinical description of the disease by Hippocrates.[91] Although it was recognized that the syndrome frequently occurred in persons suffering from chronic malaria, the difficulty in demonstrating malarial parasites in the blood during the attack of hemoglobinuria[87] led to the suspicion that blackwater fever might be caused by a separate and distinct infectious agent. It now is definitely established, however, that the syndrome occurs only in persons who have had repeated malarial infections, usually but not always of the falciparum type.

An excellent statistical study of 900 cases of blackwater fever demonstrated a definite correlation between the number of blackwater-fever cases and the number of malaria cases occurring in the same year. This correlation existed only in that portion of the population that had been exposed to malaria for one or two years. This emphasized the role of repeated malarial infections in predisposing to blackwater fever. The relation between malaria and blackwater fever is further emphasized by the similarity between the seasonal incidences of the two conditions. The maximum number of cases of blackwater fever always occurred one to two weeks after the peak incidence of falciparum malaria.[90]

Attempts to isolate a causative organism other than the malarial parasite have never succeeded. The parasites recovered from patients with blackwater fever do not appear to belong to any particular hemolytic strain, nor do they possess any unusual hemolysis-producing faculty. When blood from patients with blackwater fever was transfused into nonmalarial human subjects, typical attacks of falciparum malaria were produced, but blackwater fever never occurred.[92]

Although the great majority of cases of blackwater fever develop in persons chronically infected with *Plasmodium falciparum*,[88] *P. vivax* is occasionally responsible for an attack of hemoglobinuria. *P. malariae* infection, however, rarely or never is complicated by hemoglobinuria.[90]

The underlying etiologic relation of chronic *P. falciparum* infection and blackwater fever is definitely established, but the actual cause of the hemolysis is still obscure. Parasitization of erythrocytes is not heavy enough to account for the blood destruction on the basis of invasion of the red blood cells by parasites. Indeed, as already noted, it is usually impossible to demonstrate plasmodiums in the peripheral blood or viscera immediately before or during an attack.[87] Attacks are usually precipitated by medication with some antimalarial drug, but many cases occur spontaneously. This indicates that the antimalarial drugs in themselves are not responsible for the disease but suggests that the rapid destruction of the parasites in some way may serve to initiate the hemolytic process.

Hypersensitivity or allergy to the malaria parasite or to its breakdown products has been postulated as the cause of the hemolytic process. This theory has been advocated most enthusiastically by Fernân-Nunez,[83, 93] who cites as supporting evidence the time of development of hemoglobinuria. As a rule, the acute hemolytic attacks occur after six to twenty-four months of *P. falciparum* infection, in the period between apparent cure and relapse, at just the time that hypersensitivity or allergy might be expected to manifest itself. Surprisingly enough, extensive immunologic studies of cases of blackwater fever have not been reported, and it is impossible to state definitely whether abnormal agglutinins or hemolysins are present in cases of blackwater fever. From the nature of the hemolytic process, its sudden onset, the rapid progression and the huge numbers of erythrocytes that are destroyed, it seems likely that some agglutinin or hemolysin must be acting on the patient's erythrocytes. Investigation of cases of blackwater fever with this possibility in mind might prove informative. As previously stated, normal erythrocytes transfused into patients with blackwater fever are destroyed just as rapidly as are the patient's own erythrocytes, supporting the belief that the hemolytic factor resides in the plasma rather than in the erythrocyte. Of interest in this connection is the recent report of Maegraith, Martin, and Findlay[94] that the serums of patients with blackwater fever lack a hemolysis-inhibiting factor found in normal serum.

In spite of the huge numbers of erythrocytes that are destroyed in patients with blackwater fever, remarkably little abnormality has been detected in the red blood cells themselves. During the period of hemolysis they become very slightly spherocytic but otherwise are normal in appearance and maintain a normal resistance to hemolysis by hypotonic solutions of sodium chloride.[95] Foy and Kondi[95] observed a slight increase in susceptibility to hemolysis by lysolecithin, and Mer, Birnbaum and Kligler[96] reported that solutions of bile and bile salts hemolyzed blackwater fever cells more rapidly than normal cells. The meaning and significance of these observations are uncertain. They are the only definite changes that have been demonstrated in the erythrocytes of blackwater-fever patients, but they appear insignificant when compared with the clinical severity of the hemolytic process.

*(To be concluded)*

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# CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

## Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor**

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## CASE 31501

### PRESENTATION OF CASE

A sixty-five-year-old man entered the hospital complaining of jaundice.

About three months before admission he first noted transient attacks of mild pain in the left upper quadrant of the abdomen. These attacks increased in frequency and severity. They often came on after meals but were not attributed to any particular type of food. Three weeks before admission he noticed the onset of increasing jaundice, without pain. The stools were clay colored and more frequent than previously. During the development of the jaundice the urine was dark red. During the week before admission he was nauseated and vomited once. The appetite had been good. He had lost about 3 pounds in weight since the onset of the illness.

The past history revealed that two years before admission the patient had undergone a combined abdominoperineal resection of the rectum for carcinoma. His health after this operation had been good until the onset of the present illness. For a number of years he had had mild diabetes, which was easily controlled by small doses of insulin.

On physical examination the patient was well developed and nourished. The skin and scleras were deeply jaundiced. The eyes were slightly prominent. The pupils did not react to light; the left pupil measured 2 mm. in diameter, the right 3 mm. The heart was not enlarged. A Grade I systolic murmur was heard at the apex. Except for increased breath sounds over the right lower lobe the lungs were normal. The abdomen showed a functioning, well healed colostomy, a midline suprapubic scar and a cecostomy scar. The liver was palpable two fingerbreadths below the right costal margin, and its edge was irregular.

The temperature was 98°F., the pulse 80, and the respirations 20. The blood pressure was 130 systolic, 70 diastolic.

The urine was normal. The red-cell count was 5,100,000, with 100 per cent hemoglobin. The white-cell count was 18,400, with 80 per cent neutrophils. The nonprotein nitrogen was 41 mg. per 100 cc., and the total serum protein 5.92 gm., with 3.65 gm. of albumin and 2.27 gm. of globulin. A cephalin flocculation test was negative in twenty-four and forty-eight hours. The prothrombin time was normal. The serum bilirubin was 15.2 mg. per 100 cc. direct, and 20.5 mg. indirect. The serum phosphorus was 2.8 mg. per 100 cc., and the alkaline phosphatase 14.8 units. A blood Hinton test was negative.

An x-ray film of the chest taken on the second hospital day showed a large thorax and kyphosis of the dorsal spine. The lungs were clear, without evidence of metastatic lesions. Injection of barium through the colostomy on the seventh hospital day showed that the proximal colon contained a large amount of retained contents but no obstruction was visualized. Barium proceeded into the terminal ileum. Barium did not fill more than a small portion of the distal loop. An electrocardiogram showed normal rhythm, with a rate of 100. The axis was normal. The PR interval was 0.13 second. $T_1$ was flat, and $T_2$ and $T_3$ upright but low; the T waves in Lead $CF_2$ were upright, and those in Leads $CF_4$ and $CF_6$ inverted.

The patient was confused. He vomited frequently and received intravenous fluids. The diabetes remained under control.

On the eighth hospital day an operation was performed.

### DIFFERENTIAL DIAGNOSIS

DR. MARSHALL K. BARTLETT: As a starting point it seems to me that we can assume this is a case of obstructive jaundice. All the information points in that direction. It had been going on for three weeks and increasing fairly rapidly, so that on admission the patient had a serum bilirubin of over 15 mg. There was a history of clay-colored stools, and although there is no record of a stool examination to confirm that, I think that we must accept the history and assume that he had little, if any, bile in the intestinal tract. Attacks of pain are mentioned in the history, but they are not given much prominence and nothing is said about pain after he entered the hospital. I assume he had had no further attacks.

Here is a man who had had carcinoma of the rectum two years previously. Was the picture on entry a result of that or was it due to a new disease? If he had had enough metastatic disease to cause this marked degree of jaundice and obstruction to his biliary tree, it seems to me that he ought to have had more to show for it than he had. He had lost little weight, the red-cell count and hemoglobin were normal, and the chest film was negative, or at least showed no evidence of metastases. It seems to me unlikely that this is a picture of metastatic carcinoma from a lesion in the rectum. Of course, he could have had a small area of metastatic disease obstructing the biliary tree. I see no way to rule it out, but I think that it is unlikely. It is a well

*On leave of absence.

known hazard in these exercises to introduce a second diagnosis when one can explain the picture on the first one. In the face of that danger, my guess is that this was not a recurrence of the carcinoma.

If it was something else, what was it? The chances are that he had an obstruction either from a new tumor or from a stone. Which of those is it going to be? Here again his general condition was exceedingly good for a man with carcinoma of the head of the pancreas, carcinoma of the ampulla or carcinoma of the bile duct. He had some pain with the onset, but the nature of the attacks of pain does not help me much. The pain was on the left side, which is wrong for the gall bladder, and I do not know quite what to make of it. He had had a mild diabetes for some time. What bearing did that have on the picture? It probably had none. The incidence of gallstones in diabetic patients is slightly higher than that in normal people, which might make the statistical probability of gallstones greater, but other than that I do not believe that it had any bearing.

The laboratory data, except for revealing a normal hemoglobin and white-cell count, do not help me much. The problem, to my mind, comes down to a guess between a stone obstructing the ducts or a tumor, probably a new one if it was a tumor. Because of the fairly short duration of the illness and because of the patient's good general condition I think that the chances are a little better that in this case we are dealing with an obstructive jaundice on the basis of a stone.

Dr. Ronald C. Sniffen: Dr. Herrera performed a peritoneoscopy before operation. What did you find, Dr. Herrera?

Dr. Rodolfo E. Herrera: The main reason for performing the peritoneoscopy was to rule out metastatic carcinoma from the rectum. We believed, as Dr. Bartlett did, that this man probably had a new disease, but because we had recently had two patients with jaundice who had returned to the hospital with widespread liver metastases from carcinoma of the breast but who had not shown evidence of weight loss, a peritoneoscopy was done. In this case we found that the liver was a bit enlarged and had a greenish-yellow tinge, which was consistent with obstructive jaundice. There were no implants in the peritoneum. The gall bladder was rather large. It did not appear thickened or tense when touched with the tip of the peritoneoscope, and there were no adhesions around it to suggest that he had had previous attacks of cholecystitis. We thought that he had an obstructive jaundice, but it was impossible to tell whether he had a new growth, enlarged lymph nodes in the region of the common duct or stone. At any rate no tumor could be seen in the region of the head of the pancreas.

Dr. Sniffen: Are there pertinent x-ray films?

Dr. Clayton H. Hale: The films do not contribute much. The colon was not well prepared and shows defects that are probably fecal material. No studies of the upper gastrointestinal tract were done. The chest shows no evidence of metastatic disease.

Dr. Joseph C. Aub: How can a man who is deeply jaundiced have a normal urine? "Normal" is a dangerous word in medicine. It usually means that you do not find anything positive that you test for.

Dr. Sniffen: We made an unintentional omission in our abstract of the clinical history. The urine showed a ++ test for bile.

### Clinical Diagnosis

Obstructive jaundice.

### Dr. Bartlett's Diagnoses

Stone in common bile duct.
Obstructive jaundice.

### Anatomical Diagnoses

**Stone at bifurcation of common hepatic duct.**
Bile stasis of liver.
Aspiration pneumonia.

### Pathological Discussion

Dr. Sniffen: The operation was performed five days after the peritoneoscopy. The surgeon found a distended gall bladder containing stones and removed it. The common duct was then explored and found to be very narrow, although fine probes entered the duodenum. Fine probes could also be inserted into the right and left hepatic ducts. The head of the pancreas seemed normal. The duodenum was then opened and the ampulla visualized and palpated, but it, too, was normal. Postoperatively the patient did not do well. He was treated with intravenous fluids, transfusions and Hykinone. The bile coming from the catheter in the common hepatic duct was normal in appearance, but the average daily output was only 100 cc. His death seemed to be due in large measure to the sudden aspiration of a large amount of stomach contents.

At the time of death the patient was severely jaundiced. The operative field was in good condition, and there was gastric dilatation. The lungs were heavy; there was consolidation in both lower lobes, and gastric contents in the trachea and bronchi. The liver was dark green to brown and enlarged, weighing 2650 gm. The intrahepatic and extrahepatic bile ducts were not appreciably dilated. A rubber catheter had been inserted into the bile duct at the point of entrance of the cystic duct, and its tip lay at the bifurcation of the common hepatic duct. Scattered in the small intrahepatic bile radicles there were calculi that resembled grains of sand. Straddling the bifurcation of the common hepatic duct and lying against the tip

of the catheter there was a stone that measured 12 mm. in length and 6 mm. in diameter. It lay transversely, so that both right and left hepatic ducts were blocked. This stone was loosely held together, and when one picked it up with forceps, it was devoid of substance and felt like soft tissue. Furthermore it did not seem to be impacted in its position. I imagine that it rocked back and forth as the probe was inserted into the hepatic ducts. The kidneys were swollen and green. The tubules contained bile casts, and some of the cells of the convoluted tubules were undergoing degeneration. The interstitial tissue was edematous and infiltrated with mononuclear cells.

No metastatic tumor could be found.

## CASE 31502

### Presentation of Case

A forty-eight-year-old stockbroker entered the hospital complaining of an ulcer of the tongue.

Nine months before admission the patient noted a painless ulcer on the undersurface of the tongue near the tip. It reached a diameter of about 1 cm. and then remained stationary up to the time of admission. A blood Wassermann test was negative. Treatment with potassium iodide caused no change in the lesion. Six months before admission a painless left testicular mass was found, which had not increased in size.

The past history revealed that the patient had been a heavy user of alcohol for many years, often consuming as much as one quart of hard liquor a day. He had never had hallucinations but was troubled with tremor.

On physical examination, the patient was well developed but thin and pale. He was apprehensive. A coarse tremor shook his body, and he was unable to stand or walk. The skin and lips were dry and scaling. The tongue was beefy red, with a smooth border. It showed a coarse tremor. On the underside of the tip, to the left of the midline, was a circular, papillary, yellowish-gray lesion 1 cm. in diameter. It was well circumscribed, nontender and soft. There were no other lesions in the mouth. The cervical lymph nodes were not enlarged. Breath sounds were increased over both apices, and there was decreased resonance throughout. No rales were heard. There was a soft systolic murmur in the left second interspace. The abdomen was moderately tender in the right upper quadrant and slightly tense throughout. The left testis was enlarged to form a firm, irregular, nodular, nontender mass, measuring 4 by 2 cm. It did not transilluminate. Rectal examination revealed a soft, lobulated, slightly tender, right prostatic lobe, measuring 6 by 3.5 cm.; the left lobe was extremely firm, fixed and 2.5 cm. in diameter. The reflexes were normal.

The temperature was 101°F., the pulse 100, and the respirations 20. The blood pressure was 80 systolic, 70 diastolic.

The urine was normal except for a + test for diacetic acid. The red-cell count was 4,200,000, with 10.4 gm. of hemoglobin. The white-cell count was 5000. The nonprotein nitrogen was 13 mg. per 100 cc., and the serum chloride 80 milliequiv. per liter. The total serum protein was 5.4 gm. per 100 cc. A smear of gastric contents was negative for acid-fast bacilli.

A chest film showed mottled density throughout both lung fields, more marked in the upper than in the lower portions (Fig. 1). A bleb or cavity was present at the left apex. The diaphragm was normal in position. The costophrenic sinuses were clear. The heart and upper mediastinum were not remarkable. A flat film of the abdomen showed distention of loops of small intestine and gas in the large intestine as far as the splenic flexure. An intravenous pyelogram was unsatisfactory. The bladder was small and contracted.

During the hospital course, the patient's temperature fluctuated between 99 and 103°F., rising terminally to 106. He was disoriented and restless. He refused food and was given fluids intravenously. Lumbar puncture yielded a clear, colorless fluid. He expired on the ninth hospital day.

### Differential Diagnosis

Dr. Donald S. King: In summary, we have a forty-eight-year-old patient who was suffering from chronic alcoholism, with marked vitamin B deficiency and so-called "alcoholic neuritis." Besides that he had lesions in four organs: the tongue, the lungs, the prostate and the testis. The anemia, low serum protein, low chloride and low blood pressure can be explained largely on the basis of the alcoholic history and are not, I believe, concerned in the diagnosis.

What disease or diseases may give lesions in the four organs named? One thinks first of the old trio: syphilis, cancer and tuberculosis. The choice in this case seems easy, but it is often these apparently simple cases in which we find a catch. So let us try to apply each of these three diagnoses to the lesions as described in the tongue, lungs, prostate and testis.

The first possibility is syphilis. There was one negative Wassermann test. The patient was given potassium iodide, and the tongue lesion did not change. He could have had a gumma of the testis and associated gonococcal infection of the prostate. Syphilis of the lung can occur, but the only cases in this hospital that we believe were proved were gummas, and the x-ray appearance was quite different from that shown here. I am ready to drop syphilis from the list.

The next possibility is cancer. The description of the lesion of the tongue is not characteristic of

cancer. The adjective "papillary" is used once, but otherwise the lesion is described as an ulcer that was soft, without an indurated edge. And after nine months there were no metastases in the cervical nodes. Cancer of the tongue can, I understand, rarely metastasize to the lungs. I have never seen a case, but I doubt that the lungs would give an x-ray picture like this. And while we are considering metastatic lesions of the lung, this is a good time to point out that, if the primary cancer is in the testis, metastases to the lung usually give a snowball appearance and not such diffuse shadows as are apparent here. Metastatic malignancy from the stomach or pancreas may give an x-ray picture much like this, except that there is almost never a cavity at the left apex, which is, I believe, pictured here. So the x-ray film of the chest seems to me to be much more characteristic of another diagnosis than it is of malignancy. I am ready to dismiss cancer of the tongue and metastatic disease of the lung, but I must consider cancer again in speaking of the genitourinary tract.

That brings us to tuberculosis. The lesion of the tongue is consistent with a tuberculous ulcer. It is true that in the cases that I have seen — and they have been few — there was reasonably severe pain and tenderness. But this man was an alcoholic addict, and much of his pain may have been dissolved in alcohol. One cannot count too much on his statements. I shall therefore accept the diagnosis of a tuberculous ulcer of the tongue. The chest film, with its diffuse shadows, and what I believe to be a cavity at the left apex, seems more like tuberculosis than anything else. No sputum examination is reported, and one examination of the gastric contents is reported not to have shown acid-fast bacilli. Nevertheless, I believe that this man had pulmonary tuberculosis.

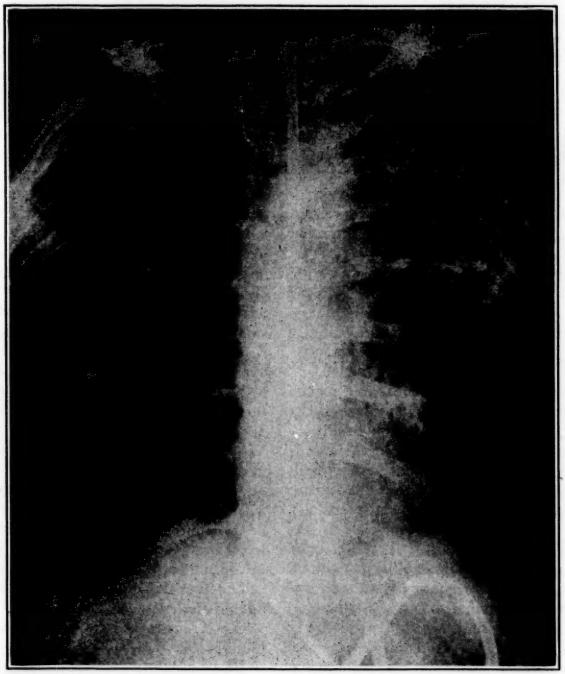

FIGURE 1.

Finally we must consider the lesions in the prostate and testis. The firm, fixed lesions described could, it seems to me, be due either to cancer or to tuberculosis. I admit that tuberculosis of the testicle without associated involvement of the epididymis is unusual, but I assume that there was at least some

involvement of the epididymis in this case. So, having accepted tuberculosis as the diagnosis for the lesions of the tongue and lungs, I shall also accept it for the prostate and testicle. There is insufficient evidence for a diagnosis of tuberculosis of the intestines or central nervous system. I shall be interested in knowing whether the small contracted urinary bladder was due to tuberculosis of the bladder wall. I shall also ask the old question whether alcoholic cirrhosis was found.

In my own study of the record I had gone this far without looking at the x-ray films of the abdomen, which were given to me yesterday. Perhaps this preview gave me information that I should not have had. But I do not believe that it influenced my diagnosis, although I am willing to grant that it confirmed my suspicions.

DR. CLAYTON H. HALE: This plain film of the abdomen shows dilated loops of small and large bowel, as described in the record. The bladder is rather small. I should say that it was characteristic of the contracted bladder of tuberculosis.

DR. KING: The two things that I noticed were a mass of calcified abdominal nodes and a rounded calcified area, which I believe is in the left testis. If the mass in the left testis is calcified, it is a strong argument for tuberculosis. Do you think that the area in question is in the left testis?

DR. HALE: It could be; I cannot be sure.

DR. RONALD C. SNIFFEN: Dr. Nathanson, you saw this patient. Would you like to comment?

DR. IRA T. NATHANSON: This man came to the Tumor Clinic with the complaint of an ulcer of the tongue, and it became apparent at the beginning that the lesion was an unusual one from the standpoint of diagnosis. The lesion appeared as described; it was papillary, yellowish gray and well circumscribed. We first thought that it was tuberculous, although it was not characteristic, as Dr. King has pointed out, of a tuberculous lesion of the tongue. Occasionally tuberculosis may be papillary or warty. The lesions that we have seen have been well circumscribed with sharp reddened edges, little or no induration and a yellowish base, and are usually tender, presumably due to secondary infection. There was surprisingly little or no induration. There were no lesions in the mouth that suggested vitamin deficiency, which occasionally causes an ulcer resembling this type of lesion. We were therefore unable to make a definite diagnosis but considered tuberculosis as one of the possibilities. The patient was put on potassium iodide to rule out the possibility of syphilis, even though the Wassermann test was negative. He was sent to the hospital for further study.

## CLINICAL DIAGNOSIS

Pulmonary tuberculosis?
Carcinoma of prostate?
Carcinoma of tongue?
Carcinoma of testicle?

## DR. KING'S DIAGNOSIS

Tuberculosis of tongue, lungs, prostate, and testes.

## ANATOMICAL DIAGNOSES

**Tuberculosis of tongue, epiglottis, larynx, lungs, testes, epididymes, prostate, bladder and left kidney.**
Mural thrombus: left ventricle.
Infarcts of spleen, right kidney and adrenal gland.

## PATHOLOGICAL DISCUSSION

DR. SNIFFEN: At the time of death the patient was quite emaciated. The ulcer on the undersurface of his tongue was 7 mm. in diameter and had a ragged border and a dirty-gray base. The mucosa of the epiglottis and larynx was thick and hemorrhagic, with areas of ulceration. Each pleural cavity contained scattered fibrous adhesions but no fluid. The lungs were heavy and riddled throughout with firm, yellowish-white nodules from pinhead size to 3 cm. in diameter. The intervening parenchyma was consolidated, and in some of the large nodules the tissue had broken down to form abscesses. Both epididymes and testes were matted together, fibrotic and riddled with abscesses; the enlargement was greater on the left than on the right. The prostate was large and almost destroyed by multiple abscesses. The left kidney contained nodules similar to those found in the lungs. All these lesions that I have described proved to be due to tuberculosis.

As an added insult there was a mural thrombus in the apex of the left ventricle. We could find no histologic reason for this; the underlying myocardium was normal, although the endocardium seemed to be destroyed at that point. Embolism, presumably from this mural thrombus, had produced infarcts in the spleen, right kidney and one adrenal gland.

The liver showed fatty change and central necrosis but no cirrhosis.

DR. R. EARLE GLENDY: How about the urinary bladder?

DR. SNIFFEN: It showed a tuberculous cystitis of the trigone where it was in contact with the prostate. There was, however, no mucosal ulceration.

DR. GLENDY: Were there ulcers in the small bowel?

DR. SNIFFEN: No.

DR. MADELAINE BROWN: Did you examine the nervous system?

DR. SNIFFEN: Yes. The brain was essentially normal, apart from an unidentified pigmentation around the substantia nigra. There were no changes indicative of pellagra.

DR. BROWN: I do not suppose that you looked at the peripheral nerves.

DR. SNIFFEN: Unfortunately, no.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

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Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

## RESIDENCIES FOR DISCHARGED MEDICAL OFFICERS

The last ten or fifteen years have witnessed a marked increase in the trend toward specialization in medicine. This has been considerably intensified by the creation of certifying boards in the many different specialties. Whether or not this trend is desirable or should be encouraged, it has had the effect of essentially adding another three or four years of postgraduate training to the already long time that it takes until physicians are prepared to enter the practice of medicine.

On the larger and better hospitals, particularly those in the big medical centers of this country and those attached to medical schools, has fallen the duty of providing this specialized training and of maintaining the high standards of medical practice that make such training possible. Under normal conditions, it has usually been possible for these hospitals to provide for a number of men equal to between 15 and 30 per cent of those who enter preliminary training as interns immediately after they receive their medical degrees. To maintain some continuity, especially in hospitals where there are short or rotating internships, it is usually necessary to choose largely from among those who received their earlier training in the same hospital. Since it is frequently desirable to inject the refreshing influence of men trained elsewhere, a small number may, under suitable circumstances, be taken from other hospitals. This often has the effect of temporarily reducing the efficiency of the hospital staff because the new persons lack acquaintance with their new environment, and in addition, it decreases the opportunities available to the men who received their early training at that institution. Furthermore, it is necessary to select men who are suited for the responsibilities entailed in the advanced positions. They not only must be able to direct the work of the interns but must also be equipped to teach both students and interns and to discharge important administrative responsibilities in the hospital.

Since the beginning of the war, men have been graduated from various medical schools at nine-month intervals and the vast majority of them have had to enter the service immediately after the completion of a nine-month internship. A few of them have had the opportunity to stay on for a second nine-month period in the so-called capacity of "resident," but actually their duties have been similar to those that constitute the latter part of the usual internship in a good hospital. An extremely small number have been permitted to stay on for a third nine-month period. Six classes of interns have already been graduated into military service in this manner, with the result that only a small percentage of these men have had more than nine months of civilian hospital training. Some of them have already been separated from military service, and most of them will be returning within the next year or two; and practically all of them will want further hospital training. In ad-

dition, many who previously had specialized training in certain fields will wish to return to similar positions because of their desire to continue in this specialty or to change to another. Perhaps a small percentage will be satisfied with a short period of refresher training before they enter or re-enter the practice of medicine.

Obviously, internships of the type that begin following graduation from medical school are not desired by the returning men. Possibly some of those who had only a short rotating service would like to have a straight internship in a teaching hospital. Those who do must, of course, compete with the men who are graduating from medical schools. It may be possible to increase to some extent the number of such internships, but it can hardly be expected that any significant number of desirable and acceptable appointments of this sort will be available to the returning servicemen.

The immediate and greatest demand will undoubtedly be for residencies, that is, for the equivalent of the second and third years of hospital training. As already indicated, such opportunities are normally offered only to one out of every five or six, or at most three, men after they have completed their first year of internship. Assuming that none of these places will be given to the men who are now finishing their internships, there will still be men equivalent in numbers to seven to nine medical-school classes who will be returning from military service within the next year or so. Should all these men apply for residencies, there would be somewhere between 21 and 54 of them competing for each place. Obviously, some expansion of opportunities can and will be arranged; but even these can hardly more than double the number of positions ordinarily available without defeating the entire purpose for which they have been or will be created.

All this bespeaks the unwiseness of the so-called "accelerated program," which was never adopted in Russia and was dropped after a short trial in Canada and Great Britain. The situation is disheartening to hospital administrators and to the chiefs of hospital services who are anxious to provide for the returning servicemen. It will appear even more discouraging to the latter, who, returning with high hopes of continuing their training, will find that the number of opportunities are even more limited, relatively speaking, than they were under normal conditions. It will require a patient understanding on the part of those who are charged with choosing the men and on the part of those who are returning and seeking these positions. Obviously, a demanding attitude on the part of the veteran, based on his service record or on his rights and privileges, can only result in bitterness and disappointment. After all, the bulk of competition for the positions that these men seek will come from men who are in exactly the same situation that they are. Those who have the not-too-pleasant task of allocating the small number of places are already embarrassed at the idea of having to turn away so many men whom they would like to take and cannot.

Each hospital will eventually work out a solution that seems best suited to its peculiar circumstances. Obviously, each institution will feel that it owes some allegiance to its own graduates, particularly since they, like others, will be unable to find positions elsewhere. Even so, they still have the broader duty and obligation to encourage only those who are most suitable by their previous training, performance and personality. All these factors must be considered, since, as previously mentioned, residents must not only care for patients but also discharge duties concerned with hospital adminis-

**MASSACHUSETTS MEDICAL SOCIETY POSTWAR LOAN FUND**

**The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:**

**George L. Schadt, *Chairman***
**Postwar Loan Fund**
**8 Fenway**
**Boston 15, Massachusetts**

tration and with the teaching of students and interns.

Most medical schools and many hospitals have already completed plans that provide for the maximum number of men. Other hospitals are still groping with the problem and trying to increase the number of opportunities and to create new and desirable ones. Still others, which prior to the war had not had residents or even interns, are contemplating the establishment of facilities that will provide training of this type. In Massachusetts, all such plans are receiving the enthusiastic support of the Committee on Postwar Planning of the Massachusetts Medical Society. It is hoped that completion of these plans will not be too long delayed and that final details will be given adequate publicity. A large number of the returning men will undoubtedly have to content themselves with some type of refresher course designed to acquaint them with the best and most modern practices in medicine. Such courses should be suitable for some men who have already had two or more years of hospital training or who were already in practice before they entered military service.

## DISABILITY DUE TO OCCUPATIONAL DERMATOSES

DISTURBANCES of the skin due to occupational factors have come to be recognized as a serious cause of disability among workmen at certain jobs. The relatively high incidence of such disturbances in general and in specific occupations is well known. The increase in the number of cases associated with war industries has been startling in certain industries. An article appearing elsewhere in this issue of the *Journal*, however, points out that little information is readily available regarding the toll that these diseases take in terms of compensation paid to the worker, the sums spent for medical treatment and hospital care or the duration of disability. The data that are available reveal how costly such conditions have become in the modern world of production and still more production. In the opinion of authorities in this field many of these losses are preventable, at least under peacetime conditions, if full advantage is taken of the principles of preventive medicine and the knowledge of corrective measures developed in the last few years, but it may take the full impact of the statistics of the end results and the costs of such disabilities to focus the requisite attention on these cases.

It is recognized, of course, that industrial accidents produce greater losses, but with occupational dermatoses accounting for 60 to 65 per cent of all occupational disease, their cost, figured in time and in dollars and cents, is a sizable one in the aggregate. It is true that many patients lose little or no time and have negligible expense, but there are numerous protracted cases and those in which the workman is unable to return to his former job, so that any stimulus to the investigation and prevention of such cases should produce benefits for both the employee and the employer. Although much has been learned about occupational dermatoses by the Office of Dermatoses Investigations of the United States Public Health Service and much has been accomplished in the way of their detection and prevention, the development of accurate statistics covering the losses resulting from such cases would without question stimulate a greater interest in them and their prevention.

## MASSACHUSETTS MEDICAL SOCIETY

### DEATHS

MOORE — George A. Moore, M.D., of Palmer, died October 6. He was in his seventy-fifth year.

Dr. Moore had been chairman of the board of trustees of the Monson State Hospital for a quarter of a century and had served as chief of staff of the Wing Memorial Hospital for the past fifteen years. He was a fellow of the American Medical Association and the American College of Surgeons and a member of the American Academy of Ophthalmology and Otolaryngology.

His widow, a sister and several nieces and nephews survive.

ZIELINSKI — Lieutenant Commander Ignatius Zielinski, U.S.N.R., formerly of Salem, died November 11 in a plane crash. He was in his forty-sixth year.

Dr. Zielinski received his degree from Tufts College Medical School in 1924. He was a surgeon on the Salem Hospital staff and had been medical examiner of the Tenth Essex district about ten years. He was a fellow of the American Medical Association. In the Navy, he first served at the dispensary of the Boston Navy Yard and later saw duty in Hawaii. He was assigned as chief surgeon to Quonset about a month ago.

His brother and five sisters survive.

## MEDICOLEGAL ABSTRACT

**Regulation of Practice by Government: Qualification to practice.** Dr. Arnold Schleifer graduated from the University of Vienna Medical School on March 4, 1938. The Illinois State Department of Registration and Education at that time recog-

nized the Vienna school as reputable and in good standing. Four years later it retroactively suspended recognition of the university, thus purporting to deprive Dr. Schleifer of the right to be examined to practice medicine in Illinois.

Dr. Schleifer brought mandamus to compel the director and the superintendent and others of the department to grant him access to the examination of candidates to practice medicine in all its branches. An order denying this petition was entered, and Dr. Schleifer appealed. The appeal was allowed on the ground that the ruling of the department was not "fair, reasonable and impartial." — (*Schleifer* v. *Department of Registration and Education*, N. E. [2d] 398, Appellate Court of Illinois, First Dist., May 29, 1945.)

## MISCELLANY

### DINNER IN HONOR OF DR. MINOT

Dr. George Richards Minot, Nobel Laureate in Physiology and Medicine 1934, professor of medicine, Harvard Medical School and director of the Thorndike Memorial Laboratory of the Boston City Hospital, reached his sixtieth birthday on December 2. A testimonial dinner was tendered him in the Aesculapian Room of the Harvard Club of Boston on December 5 by some of his intimate associates and scientific colleagues. Dr. William B. Castle served as toastmaster, and the speakers included Drs. Henry A. Christian, C. Sidney Burwell, James W. Manary, Laurence B. Ellis, Elliott P. Joslin, Reginald Fitz, J. H. Means and Francis M. Rackemann. The speakers reviewed some of the outstanding personal characteristics and scientific contributions of the guest of honor. Dr. Reginald Fitz, representing the president and House of Delegates of the American Medical Association, took the occasion to award to Dr. Minot its Distinguished Service Medal for his outstanding achievements. Dr. Castle presented to Dr. Minot a bound volume of letters of greeting from numerous scientists throughout the world who have made notable contributions to hematology and nutrition.

### COLONEL PARSONS AWARDED LEGION OF MERIT

Lieutenant Colonel C. Langdon Parsons, M.C., A.U.S., on leave of absence as a member of the Surgical Staff of the Massachusetts General Hospital and as assistant in surgery at the Harvard Medical School, was recently awarded the Legion of Merit for meritorious service as chief of the surgical service of a station hospital that handled the majority of cases of maxillofacial injury in the Mediterranean theater from April 1, 1944, to April 1, 1945.

## NOTICES

### ANNOUNCEMENTS

Dr. William Dameshek announces the removal of his office to 192 Beacon Street, Boston.

Dr. Karl D. Kasparian announces the opening of his office at 395 Commonwealth Avenue, Boston, for the practice of general surgery.

Dr. Leo Maletz announces the opening of his office at 80 Nahant Street, Lynn, for the practice of psychiatry and neurology.

Dr. Joseph D. Wassersug, formerly of the Norfolk County Hospital, announces the opening of his office at 1159 Hancock Street, Quincy, for the practice of internal medicine.

### BOSTON SOCIETY OF BIOLOGISTS

A meeting of the Boston Society of Biologists will be held in the Surgical Amphitheater (3A) of the White Building, Massachusetts General Hospital, on Wednesday, December 19, at 8 p.m.

PROGRAM

Naphthoquinone Antimalarials. Dr. Louis F. Fieser.
A Fibrous Modification of Insulin. Dr. David Waugh.
Streptomycin. Dr. Donald C. Anderson.
Sulfathiadiazole. Drs. Manson Meads and Maxwell Finland.

### HEALTH EDUCATION INSTITUTE

An Institute in Health Education, sponsored by the Massachusetts Tuberculosis League, is to be held at Simmons College, Boston, from February 4 through March 29. The first five weeks of this institute will be devoted to lectures and discussions by local and national experts, and the remaining three weeks to supervised field work.

The primary purpose of the course is to train assistants in health education to work under supervision in the tuberculosis associations of Massachusetts. The training program, however, will also be open to any educational workers in the tuberculosis field desiring a brush-up course. Health workers in other voluntary agencies and in the official agencies of Massachusetts are also invited to attend all or part of the Institute.

Inquiries regarding the detailed program and enrollment in the Institute should be addressed to the Massachusetts Tuberculosis League, 1148 Little Building, Boston 16.

### CUTTER LECTURE

The Cutter Lecture on Preventive Medicine will be given by Dr. Donald Hunter, F.R.C.P., physician to the London Hospital, London, England, at 5 p.m. on Monday, December 17, in the amphitheater of Building D of the Harvard Medical School. His subject will be "Research in Industrial Medicine in Great Britain at War."

The medical profession, medical and public-health students, the press and others interested are cordially invited to attend.

## SOCIETY MEETINGS AND CONFERENCES

### CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, DECEMBER 20

FRIDAY, DECEMBER 21

*9:00-10:00 a.m. The Clinical Evaluation of an Anti-Hemophilic Fraction of Plasma. Dr. Louis K. Diamond. Joseph H. Pratt Diagnostic Hospital.
*9:00-10:00 a.m. Medical clinic. Isolation Amphitheater, Children's Hospital.
*10:00 a.m.-12:00 m. Medical staff rounds. Peter Bent Brigham Hospital.
10:50 a.m. Squamous Eruptions. Dr. George Schwartz. (Postgraduate clinic in dermatology and syphilology.) Amphitheater, Dowling Building. Boston City Hospital.

MONDAY, DECEMBER 24

*12:00 m.-1:00 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

WEDNESDAY, DECEMBER 26

*12:00 m. Clinicopathological conference. Children's Hospital.

*Open to the medical profession.

DECEMBER 17. Cutter Lecture. Notice above.

DECEMBER 18. South End Medical Club. Page 710, issue of December 6.

DECEMBER 19. Boston Society of Biologists. Notice above.

JANUARY 7-APRIL 22, 1946. Metropolitan State Hospital. Eleventh postgraduate seminar in neurology and psychiatry. Page 314, issue of September 6.

JANUARY 10. Fatigue, Its Significance and Treatment. Dr. Frank N. Allan. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

FEBRUARY 2. American Board of Obstetrics and Gynecology. Page 514, issue of October 25.

FEBRUARY 4-MARCH 29. Health Education Institute. Notice above.

### DISTRICT MEDICAL SOCIETY

WORCESTER

JANUARY 9. St. Vincent Hospital.
FEBRUARY 13. Worcester State Hospital.
MARCH 13. Worcester Memorial Hospital.
APRIL 10. Hahnemann Hospital.
MAY 8. Annual meeting.